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## FACIAL GROWTH IN RELATION TO DENTAL OCCLUSION*

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THE problem of growth of face and head has received considerable attention, but this preoccupation has been, with a few exceptions, without reference to the type of dental occlusion manifested by the subjects. Franke,[1] Brash,[2, 3] Goldstein and Stanton,[4] and others have taken cognizance of dental occlusion in studies on growth of particular regions of the face, primarily the alveolar arches. Only Hellman,[5, 6, 7] and Smyth and Young,[8] however, have presented evidence, to our knowledge, which has considered growth of the whole head and face as related to dental occlusion.

Hellman's pioneer work on face growth is or should be well known. His material, however, is presented primarily without regard to dental occlusion; that is, the data include cases with normal occlusion and malocclusion combined. Nevertheless, in one excellent monograph[9] he considers the face of subjects manifesting Angle's Class III malocclusion,† but the evidence pertains to conditions in the adult, albeit pertinent comment is made on the probable influence of growth. Two other papers[10, 11] from his prolific pen deal with the face as influenced, respectively, by Class II and open-bite (Class I) malocclusions, again, however, especially as manifest in the adult.

The material collected by Smyth and interpreted by Young was, on the other hand, rigidly selected on the basis of excellent dental occlusion; moreover the age range in this excellent study was also limited to the period 2 to 14 years.

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†The Class I, Class II, and Class III used in the following in describing kind of occlusion refer to the Angle classification of malocclusion. Class I has reference to all disturbances in the dental alignment in which the mesiodistal relationship of the maxillary mandibular side teeth (cuspid and teeth posterior thereto) is not involved or is involved to a limited extent; Class II, the mandibular side teeth are in distal relationship to the corresponding maxillary teeth; Class III, the mandibular side teeth are in mesial relationship to the corresponding maxillary teeth.

In short, there seemed to exist a definite need for additional work in this field, in which the correlation of growth in the head and the face with type of dental occlusion should be considered extensively and in detail. The present contribution is an attempt in this direction.

## MATERIAL AND METHOD

The subjects are males only, and in order to attain some homogeneity of stock, Jewish boys, with relatively few exceptions (8 per cent), were selected throughout. Practically all of the subjects were native American stock (two or three were foreign born, but reared from infancy in the United States). In another paper[12] it has been shown that the non-Jewish boys in the present instance were quite like the Jewish in their physical measurements.

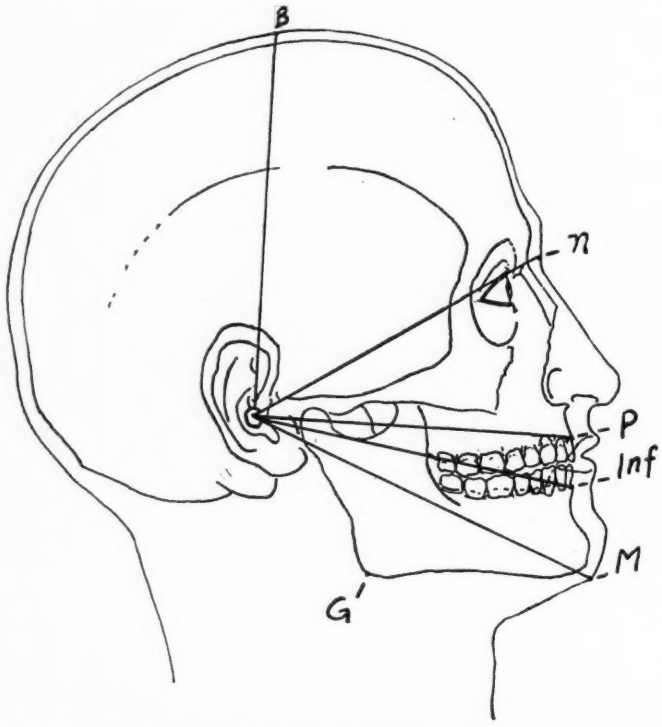


Fig. 1.—Landmarks demarcating the various characters measured. (*B*, bregma; *N*, nasion; *P*, prosthion; *Inf*, infradentale; *M*, menton; *G*, gonion.)

It would have been desirable to limit the study to one economic group; for Bakwin,[13] Boas,[14] and others have clearly shown that growth is appreciably affected by nutritional and environmental factors. However, regretfully enough, circumstances did not permit such refinement of method. The group as a whole was of lower middle class and seemed fairly well nourished except, perhaps, a group of children (7, 9, and 11 years) who came from the lower East Side of New York.

All measurements were taken by the first of the authors; the classification of occlusion was made by the second author; the interpretation is the joint responsibility of both authors. The instruments used were the usual sliding calipers, a slightly modified Hrdlička spreading calipers (lighter in weight than

the original kind and made in the machine shop of New York University), and the Todd head-spanner. Each instrument was repeatedly checked as to accuracy. The landmarks (see Fig. 1) and methodology were strictly according to the International Agreement as interpreted by Dr. Hrdlička.[15] Depth diameters were measured with the head-spanner, essentially according to the method practiced by Hellman, the only difference being that the scale was calibrated to read from the center of the horizontal rods of the instrument instead of from the top, so as to have all radial measurements read from one axis, approximately the middle of the ear holes. A diameter which is new, so far as we are aware, is the nasion-gonion, taken on the left side with teeth of subject normally closed. It is used in bringing the menton-gonion depth diameter into the sagittal plane when plotting the profile of the face. The manner in which the profile of the face was plotted will be discussed in detail later.

The measurements of menton-gonion and ramus height given herein are averages of observations taken on the left and the right sides of the face.

## AGE

The chronologic age range of the subjects was 2½ to 3½ years to and including 20½ to 21½ years, taken at biannual intervals; for example, 4½ to 5½ years would follow the first group, 6½ to 7½ years thereafter, and so on. Fifty subjects were examined in each age interval. A group of 50 old men of an average age of 74 years was also included in order to note the effects of senility.

In segregating the data with regard to occlusion, the dental age essentially according to Hellman's criteria was used instead of chronologic age. Only in this manner of organization was it possible to attain fairly presentable series after dividing the available material according to dental normal and abnormal occlusion, respectively.

TABLE I

AVERAGE AGE AND STANDARD DEVIATION OF DENTITION STAGES IN CASES WITH NORMAL AND ABNORMAL OCCLUSION, RESPECTIVELY

| HELLMAN CLASS | PRESENT DATA | | | | | | N. Y. U. CLINIC | | | | | | HELLMAN'S DATA ABNORMAL (?) | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | NORMAL | | | ABNORMAL | | | NORMAL | | | ABNORMAL | | | | | |
| | | MEAN | S. D. | | MEAN | S. D. | | MEAN | S. D. | | MEAN | S. D. | | MEAN | S. D. |
| IIA | (37) | 4.41 | 1.13 | ( 58) | 3.94 | 1.72 | (98) | 4.35 | 1.06 | ( 86) | 4.14 | 1.07 | ( 17) | 5.53 | 1.52 |
| IIIA | (22) | 8.14 | 1.74 | (104) | 8.88 | 1.51 | (28) | 7.37 | 0.99 | (113) | 8.28 | 1.32 | (115) | 9.00 | 1.41 |
| IIIC | ( 4) | 13.06 | | ( 21) | 11.92 | 1.65 | | | | | | | ( 56) | 12.70 | 1.25 |
| IVA | (44) | 15.7 | 2.44 | (116) | 15.36 | 2.46 | | | | | | | (156) | 14.78 | 2.07 |
| IVC | ( 5) | 19.25 | | ( 29) | 18.59 | 1.84 | | | | | | | ( 21) | 18.62 | 2.36 |
| VA | (14) | 19.5 | | ( 34) | 19.58 | 1.27 | | | | | | | ( 29) | 20.83 | 2.07 |

NOTE.—Figures in parentheses refer to number of cases. Significance of each class is as follows:

IIA = presence of at least 3 deciduous second molars.
IIIA = presence of at least 3 permanent first molars.
IIIC = beginning of eruption of permanent second molars (eruption through gums of any one or two, regardless whether maxillary or mandibular).
IVA = presence of at least 3 permanent second molars.
IVC = beginning of eruption of third molars.
VA = presence of at least 3 third molars.

Table I presents the average age of each Hellman dentition stage according to his own published data,[6] and as occurring in normal and abnormal occlu-

sion, respectively, in the present study and in another group of children reported upon by us.[16] When number of cases justified the measure, variability in terms of standard deviation was determined.

Some disagreement between Hellman's and our results is immediately perceptible. It is to be recalled, however, that Hellman's series are a combined normal and abnormal occlusion group; too, many of his subjects were probably private orthodontic patients, which, as regards age, may involve a factor of selection. Indeed, inasmuch as every other year instead of an annual interval was utilized in our own study, there is here too an element of selection relative to average age and time of tooth eruption. There can be little doubt that many more data are needed in order to establish reliable age means for the several dentition stages. As a matter of fact, these may vary in different ethnic or even economic groups.

Returning to the differences in time of tooth eruption in normal occlusion and malocclusion, as indicated in Table I, we note with interest that both in the present study and in the New York University Clinic group, the eruption of the whole deciduous dentition occurs somewhat earlier in abnormal than in normal occlusion, and conversely, the permanent first molar appears earlier in normal than in abnormal occlusion. The permanent second molars, however, also appear earlier, on the whole, in cases with abnormal occlusion. Practically no difference is evident in the time of third molar eruption with reference to type of occlusion, except apparently at the beginning of eruption (stage IVC) when normal occlusion is represented by too few cases (5) to make this difference reliable.

The meaning of these interesting differences in time of eruption of the teeth is not clear. Our feeling in the matter is that the question might well be left open, for reasons already indicated above, until additional corroborative data on this phase of the problem become available.

### MEAN DIMENSIONS IN CASES WITH NORMAL OCCLUSION AND MALOCCLUSION, RESPECTIVELY

The analysis of the data will first proceed in the orthodox manner of comparing all cases with normal occlusion with all those manifesting malocclusion. The distinction between normal and abnormal occlusion was that usually made by the practicing orthodontist: normal occlusion referred to dentitions in which the "cheek" teeth were anatomically in correct occlusion and the surface-to-surface contact of the incisors was not, or only slightly, broken; any appreciable deviation from these conditions signified abnormal or malocclusion. Reference may be made to a previous publication[17] in which the general question of classification of dental occlusion is treated in detail.

An interesting paragraph culled from a paper by Chapman[18] has this to say about occlusion and head growth:

"A knowledge of dentition in the study of growth of the jaws cannot be overestimated, because, within limits, one is able to diagnose if the dentition, i.e., the occlusion, is normal; and if it is normal, there are good grounds for

assuming that the development of the masticatory face* is normal; and if that is normal, it may be presumed that the head also is normal."

How a "normal" face or head would be distinguished from an "abnormal" one is not clear; nor indeed are the "good grounds" for the presumption of a correlation, as implied, between type of occlusion and size and form of head and face. There may be such a correlation especially as concerns specific types of malocclusion (e.g., Angle's Class III, open-bite of incisors, etc.), but actually little quantitative work has been done, as already indicated, either to corroborate or to disprove this view. But let us to our material to check upon this very point; namely, if and how the head and face of individuals with normal dental occlusion differ from the head and face of individuals with abnormal dental occlusion.

## THE HEAD

The mean dimensions of the head with reference to normal and abnormal occlusion are given in Table II. Maximum length, we note, is remarkably alike in both normal and abnormal occlusion except at stages IIIC and IVC when the head seems appreciably longer in cases with abnormal occlusion. But the "normal" group is represented by very few numbers at these dentition stages, and the differences may easily be due to this factor.

TABLE II

MEAN DIMENSIONS IN CASES WITH NORMAL AND ABNORMAL OCCLUSION: THE HEAD

| HELLMAN CLASS | LENGTH | | WIDTH | | HEIGHT | | MINIM. FRONTAL | |
|---|---|---|---|---|---|---|---|---|
| | N | AB | N | AB | N | AB | N | AB |
| IIA | 170.8* | 170.2 | 140.6 | 135.7 | 118.3 | 117.5 | 96.0 | 92.5 |
| IIIA | 178.1 | 179.6 | 146.0 | 146.6 | 121.8 | 122.5† | 100.0 | 100.3 |
| IIIC | 182.0 | 184.8 | 150.0 | 149.4 | 123.5 | 126.2 | 99.0 | 101.6 |
| IVA | 190.6 | 190.2 | 154.8 | 153.7 | 127.0 | 126.3 | 103.3 | 102.7 |
| IVC | 187.0 | 194.4 | 154.0 | 156.0 | 126.0 | 128.2 | 100.0 | 104.6 |
| VA | 195.0 | 195.0 | 156.0 | 156.1 | 125.0 | 126.6 | 104.0 | 102.9 |

NOTE.—For number of cases involved in each category see Table I.
*Based on 36 cases.
†Based on 103 cases.

Maximum width of head, except at stage IIC, appears also substantially the same regardless of type of occlusion. Wherefore an appreciably narrower head in the small children with abnormal occlusion, as indeed is the case in their minimum frontal region, is not altogether clear, although we shall have occasion to refer back to this condition later in our discussion.

Height of head from porion to bregma is likewise much the same in both the normal and the abnormal categories. The differences occurring are slight and not constant.

The minimum frontal dimension is also very similar in normal and abnormal occlusion except at stage IIA when, as already noted, the forehead appears narrower on the whole in cases with abnormal occlusion. At stages IIIC and IVC also the discrepancies are appreciable and in favor of abnormal occlusion. As indicated above, however, the poor representation of normal occlusion at these stages vitiates their comparative value.

*According to Chapman the "masticatory face is the area below the supra-glabellar depression (often imperfectly marked and so in practice the nasion is used) and in front of the external auditory meatuses."

On the whole, it seems to us that there is no material difference in the size of the head, except perhaps the width in early childhood, in individuals segregated according to normal and abnormal dental occlusion, respectively.

### FACE WIDTHS

Table III considers the relationship of dental occlusion with the widths of the face. Width of the upper face (bizygomatic) at stage IIA does appear tangibly narrower in abnormal occlusion, indeed, just as was noted in width of head at this time. The distance between the condyles (bicondylar width) likewise is appreciably narrower in abnormal occlusion, especially at stages IIA and VA. However, scarcely one millimeter differentiates (and in no constant manner) the bigonial or nose widths in normal and abnormal occlusion. Mouth width, it may be noted, tends to be smaller in malocclusion cases.

TABLE III

MEAN DIMENSIONS IN CASES WITH NORMAL AND ABNORMAL OCCLUSION: FACE WIDTHS

| HELLMAN CLASS | BIZYGOMATIC | | BICONDYLAR | | BIGONIAL | | NOSE | | MOUTH | |
|---|---|---|---|---|---|---|---|---|---|---|
| | N | AB | N | AB | N | AB | N | AB | N | AB |
| IIA | 114.5 | 109.8 | 105.0 | 100.3 | 80.1 | 78.8 | 27.6 | 27.6 | 39.5 | 38.3 |
| IIIA | 124.4 | 125.2 | 114.5 | 113.6 | 87.4 | 88.9 | 30.0 | 30.4 | 45.0 | 45.1 |
| IIIC | 130.0 | 130.4 | 119.0 | 119.5 | 92.0 | 92.8 | 33.5 | 32.4 | 47.0 | 48.1 |
| IVA | 137.1 | 136.3 | 127.9 | 126.7 | 99.3 | 98.4 | 35.1 | 34.5 | 50.0 | 49.6 |
| IVC | 139.0 | 141.2 | 132.0 | 132.1 | 103.0 | 102.6 | 35.0 | 35.2 | 53.0 | 50.9 |
| VA | 142.0 | 140.8 | 134.0 | 130.5 | 101.0 | 101.9 | 36.0 | 36.2 | 54.0 | 52.1 |

NOTE.—For number of cases in each category see Table I.

In short, the evidence points to some relationship between face width and dental occlusion, the face on the average being narrower in abnormal than in normal occlusion.

### FACE LENGTHS

Turning to length of face (Table IV) we note that definitely at stage IIIA and possibly at IVC the total face height (nasion-menton) is greater in abnormal occlusion. Indeed, this tendency toward greater length in abnormal occlusion is, at all the dentition stages except IIA, also evident in the upper face height (nasion-prosthion) and in the nose. On the other hand, lower face height

TABLE IV

MEAN DIMENSIONS IN CASES WITH NORMAL AND ABNORMAL OCCLUSION: FACE LENGTHS

| HELLMAN CLASS | NASION-MENTON | | NASION-PROSTHION | | INFRAD.-MENTON | | RAMUS | | NOSE | |
|---|---|---|---|---|---|---|---|---|---|---|
| | N | AB | N | AB | N | AB | N | AB | N | AB |
| IIA | 91.1 | 90.5* | 56.6 | 55.6 | 30.6 | 30.4 | 40.8 | 40.9 | 40.2 | 39.7 |
| IIIA | 99.1 | 103.2 | 62.6 | 65.1 | 32.0 | 32.6 | 45.4 | 45.9 | 46.5 | 48.1 |
| IIIC | 110.0 | 109.3 | 68.0 | 68.8 | 36.0 | 34.8 | 50.7 | 48.7 | 51.0 | 51.1 |
| IVA | 115.9 | 115.7 | 70.5 | 71.5 | 38.2 | 37.6 | 57.9 | 55.6 | 53.4 | 53.6 |
| IVC | 114.0 | 121.8 | 69.0 | 73.7 | 39.0 | 40.1 | 55.0 | 59.6 | 53.0 | 55.9 |
| VA | 121.0 | 121.7 | 73.0 | 74.4 | 41.0 | 40.0 | 59.0 | 56.7 | 57.0 | 58.1 |

NOTE.—For number of cases in each category see Table I.
*Based on 57 cases.

(infradentale-menton) manifests little difference with respect to occlusion. Height of ramus also appears much the same regardless of occlusion. Mention

should be made that, as a result of the unavoidable difficulty in accurately measuring ramus height, extreme caution must be exercised in the interpretation of this diameter.

### FACE DEPTH

Depth of face is considered in Table V. A fairly clear tendency is discernible for depth, at all levels of the face, to be greater in normal than in abnormal occlusion, except at stage IIIA and possibly stage IVC when the reverse is apparent.

TABLE V

MEAN DIMENSIONS IN CASES WITH NORMAL AND ABNORMAL OCCLUSION: FACE DEPTHS

| HELLMAN CLASS | AURICULO-NASION | | AURICULO-PROSTHION | | AURICULO-INFRA-DENTALE | | AURICULO-MENTON | | MENTON-GONION | | NASION-GONION | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | N | AB | N | AB | N | AB | N | AB | N | AB | N | AB |
| IIA | 81.8 | 81.2* | 78.2† | 78.4‡ | 77.1§ | 75.8¶ | 85.8† | 83.1‡ | 70.9 | 69.8 | 100.0 | 97.9 |
| IIIA | 86.6 | 88.3 | 86.0 | 88.4 | 85.4 | 86.0 | 95.7 | 97.7 | 78.8 | 80.6 | 108.9 | 110.4 |
| IIIC | 92.5 | 92.0 | 91.5 | 90.4 | 89.7 | 90.4 | 103.7 | 102.2 | 89.7 | 86.7 | 118.0 | 116.5 |
| IVA | 96.8 | 96.6 | 95.8 | 93.6 | 95.9 | 93.3 | 110.8 | 108.4 | 97.0 | 96.2 | 125.9 | 124.4 |
| IVC | 95.0 | 99.1 | 93.0 | 98.1 | 94.0 | 99.2 | 112.0 | 114.8 | 98.0 | 99.6 | 123.0 | 129.7 |
| VA | 101.0 | 99.8 | 102.0 | 98.8 | 103.0 | 100.2 | 119.0 | 117.1 | 100.0 | 99.0 | 131.0 | 129.3 |

NOTE.—For number of cases involved in each category see Table I.
*Based on 57 cases.
†Based on 36 cases.
‡Based on 56 cases.
§Based on 33 cases.
¶Based on 55 cases.

### SIGNIFICANCE OF DIFFERENCES

It may be well at this point to question the significance of the differences between normal and abnormal occlusion, noted in the preceding. May not these differences be due merely to a discrepancy in chronologic age? For example, the mean age of the *normal* occlusion group at stage IIA is almost a half year *greater* than that of the *abnormal* group; at stage IIIA, on the other hand, the *abnormal* group is *older* by even more than a half year than the *normal* occlusion group. In fact, there is a corresponding tendency for the various dimensions at stage IIA to be greater in normal than in abnormal occlusion, and at stage IIIA to be somewhat greater in abnormal than in normal occlusion. But this is a trend only, a number of characters in both these stages manifesting little or no difference despite the discrepancy in chronologic age. Normal and abnormal occlusion are alike in chronologic age at dentition stage VA; yet appreciable differences in certain characters are plainly evident.

We are inclined to believe that the effect of growth of the face on occlusion, and vice versa, where indicated in the preceding, is real, despite the disparity of chronologic age in normal and abnormal occlusion at certain stages. Reason for this belief will be more clearly discernible later when abnormal occlusion is broken up into specific types. More will be said in our Discussion, however, with regard to chronologic age and dentition stage as measures of time in the study of growth.

Another legitimate question is: Are the differences noted sufficiently large to be significant, or are they a result of chance and probably would disappear

if other, different, groups of individuals were examined? The question is obviously pertinent, and observations on other comparable groups of subjects would be the ideal check. Until such a check is forthcoming, however, we have had recourse to a standard statistical method which treats of this very question. This method, in brief, is to the effect that when the difference between the means is less than four times $\sqrt{a^2 + b^2}$, where $a$ and $b$ equal the probable error of the first and second means, respectively, the difference is not significant. In other words, if a figure in the last columns in Table VI (stages IIA and IVA, respectively) is less than four, the probability is that the difference between the averages compared is not significant, while if it is four or over, the probability is very great that the difference is real and would occur in another sample or group of individuals. Table VI also gives the probable error (P.E.) of the averages considered and the various steps by which the pertinent figures in the last columns were reached.

TABLE VI

PROBABLE ERROR (P.E.) OF MEANS AND DIFFERENCES BETWEEN THE MEANS AND THE RESULTANT STATISTICAL SIGNIFICANCE OF THESE DIFFERENCES AT DENTITION STAGES IIA AND IVA

| DIFF./ CHARACTER | STAGE IIA | | | | | STAGE IVA | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | P. E. OF MEANS | | DIFF.* OF MEANS | P. E. OF DIFF. | DIFF. / P. E. OF DIFF. | P. E. OF MEANS | | DIFF. OF MEANS | P. E. OF DIFF. | DIFF. / P. E. OF DIFF. |
| | NORM. | AB. | | | | NORM. | AB. | | | |
| *Head:* | | | | | | | | | | |
| Length | 0.77 | 0.59 | +0.6 | 0.97 | 0.62 | 0.55 | 0.46 | 0.0 | 0.72 | 0.0 |
| Width | 0.62 | 0.42 | +4.9 | 0.75 | 6.53 | 0.50 | 0.30 | 0.0 | 0.58 | 0.0 |
| Height | 0.45 | 0.36 | +0.8 | 0.58 | 1.38 | 0.48 | 0.31 | −1.6 | 0.57 | 2.8 |
| Min. Frontal | 0.62 | 0.32 | +3.5 | 0.69 | 5.07 | 0.41 | 0.27 | +1.1 | 0.49 | 2.2 |
| *Face Widths:* | | | | | | | | | | |
| Bizygomatic | 0.74 | 0.49 | +4.7 | 0.88 | 5.34 | 0.48 | 0.35 | +1.1 | 0.59 | 1.86 |
| Bicondylar | 0.82 | 0.56 | +4.7 | 0.99 | 4.75 | 0.52 | 0.37 | +3.5 | 0.64 | 5.47 |
| Bigonial | 0.61 | 0.38 | +1.3 | 0.72 | 1.81 | 0.49 | 0.38 | −0.9 | 0.62 | 1.45 |
| Nose | 0.23 | 0.20 | 0.0 | 0.30 | 0.00 | 0.26 | 0.17 | −0.2 | 0.31 | 0.6 |
| Mouth | 0.40 | 0.25 | +1.2 | 0.47 | 2.55 | 0.32 | 0.20 | +1.9 | 0.38 | 5.00 |
| *Lengths:* | | | | | | | | | | |
| Nasion-Menton | 0.59 | 0.46 | +0.6 | 0.75 | 0.80 | 0.64 | 0.43 | −0.7 | 0.77 | 0.9 |
| Nasion-Prosth. | 0.47 | 0.40 | +1.0 | 0.62 | 1.61 | 0.44 | 0.28 | −1.4 | 0.52 | 2.7 |
| Infradentale | 0.23 | 0.17 | +0.2 | 0.28 | 0.71 | 0.29 | 0.19 | +1.0 | 0.35 | 2.9 |
| Ramus | 0.34 | 0.33 | −0.1 | 0.47 | 0.21 | 0.71 | 0.39 | +2.3 | 0.81 | 2.8 |
| Nose | 0.38 | 0.28 | +0.5 | 0.47 | 1.06 | 0.44 | 0.24 | −1.1 | 0.50 | 2.2 |
| *Depths:* | | | | | | | | | | |
| Auric.-Nasion | 0.32 | 0.37 | +0.6 | 0.49 | 1.22 | 0.39 | 0.29 | +1.2 | 0.49 | 2.4 |
| Auric.-Prosth. | 0.46 | 0.44 | −0.2 | 0.64 | 0.31 | 0.46 | 0.34 | +3.2 | 0.57 | 5.6 |
| Auric.-Infrad. | 0.50 | 0.46 | +1.3 | 0.68 | 1.90 | 0.50 | 0.38 | +2.8 | 0.63 | 4.4 |
| Auric.-Menton | 0.69 | 0.51 | +2.7 | 0.86 | 3.14 | 0.66 | 0.44 | +1.9 | 0.79 | 2.4 |
| Menton-Gonion | 0.47 | 0.38 | +1.1 | 0.60 | 1.83 | 0.53 | 0.32 | +1.0 | 0.62 | 1.6 |
| Nasion-Gonion | 0.54 | 0.42 | +2.1 | 0.69 | 3.04 | 0.59 | 0.39 | +1.7 | 0.63 | 2.7 |

*Normal minus Abnormal: A plus sign indicates a greater normal average; a minus sign a greater abnormal dimension.

The dentition stages IIA and IVA only were utilized in this analysis, since these especially are represented by fairly adequate numbers both in normal and in abnormal occlusion. These particular stages, moreover, represented two quite distinct periods of development, dental and physical: IIA signifying the com-

pleted deciduous dentition in early childhood, whereas IVA represents the full permanent dentition (second molars) in late adolescence.

Turning to the results in Table VI, and first as occurring at dentition stage IIA or early childhood, we note the following. The narrower head and forehead in abnormal occlusion is in each instance statistically significant. Width of the upper face, too, is significantly narrower in abnormal occlusion. No statistically significant difference between normal and abnormal occlusion is evident in face length. Except for the depth of the mandible (menton-gonion) and perhaps the auriculo-menton dimension which manifests a difference approximating statistical significance, there is no significant relationship at this stage between depth of face and normal or abnormal occlusion.

Regarding stage IVA, or adolescence, we note remarkable changes. The significant differences in head width and minimum frontal dimension, noted in infancy, are now not evident, having apparently become eliminated in the course of growth. Mouth width is now significantly narrower in abnormal occlusion, and the bicondylar distance still is considerably narrower in abnormal occlusion. Maximum width of face (bizygomatic), on the other hand, is now also not significantly different in normal or abnormal occlusion. Differences in upper and lower face lengths tend toward significance. And depth of face in normal and abnormal occlusion now manifests either an approach to significant differentiation, or the greater depth from the ear to the mouth region in normal occlusion is definitely significant.

In brief, there is a suggestion, to say the least, that in early childhood the chief difference in the head and face of cases with normal occlusion in contrast to subjects with malocclusion is greater width in the former. During the process of growth these differences in width of head, and even to some extent in width of face, become eliminated, while length of face and especially auricular depth to the mouth region become more differentiated, the face becoming longer in height and shorter in depth in subjects manifesting abnormal occlusion.

## FACIAL PROFILES

The profile of the face can readily be projected with the aid of the data presented herein. Hellman, to our knowledge, was the first to plot such profiles from measurements on the living. His use of these profiles is well known and comprises a real contribution to methodology in this field. Our own method of projecting the profile of the face is essentially like that employed by Hellman except for one important addition.

It is to be recalled that the facial projection is in the median sagittal plane. The whole figure can be directly plotted in this plane except the menton-gonion dimension or depth of the lower jaw. This latter diameter is brought into the sagittal plane thus: the bigonial dimension is drawn, and from each end of the latter, with a compass opened to the extent of the menton-gonion dimension, are plotted arcs which cross. The distance between the intersection of the arcs and the middle of the bigonial line is the diameter which, with one end of the compass on the menton point, would make an arc whereupon the gonion point must fall in the sagittal plane. So far Hellman and we agree, but Hellman apparently now locates the gonion on the arc just mentioned according to the

height of the ramus. In any event, a more accurate method of locating gonion on the menton-gonion arc, we think, is by means of the nasion-gonion control. This latter dimension is brought into the sagittal plane precisely in the same manner as the menton-gonion dimension described above. Hence, the arcs respectively projected by menton-gonion (from menton) and nasion-gonion (from nasion) will meet at a point which must needs be a very close approximation to the position of gonion in the median sagittal plane.

Fig. 2 represents the profile of the face, at each dentition stage, of individuals with normal and abnormal occlusion, respectively. Hellman's data have also been projected for comparative purposes. The auriculo-nasion diameter is at the same angle in every instance, to a degree which places the face in approximately the Frankfort plane.

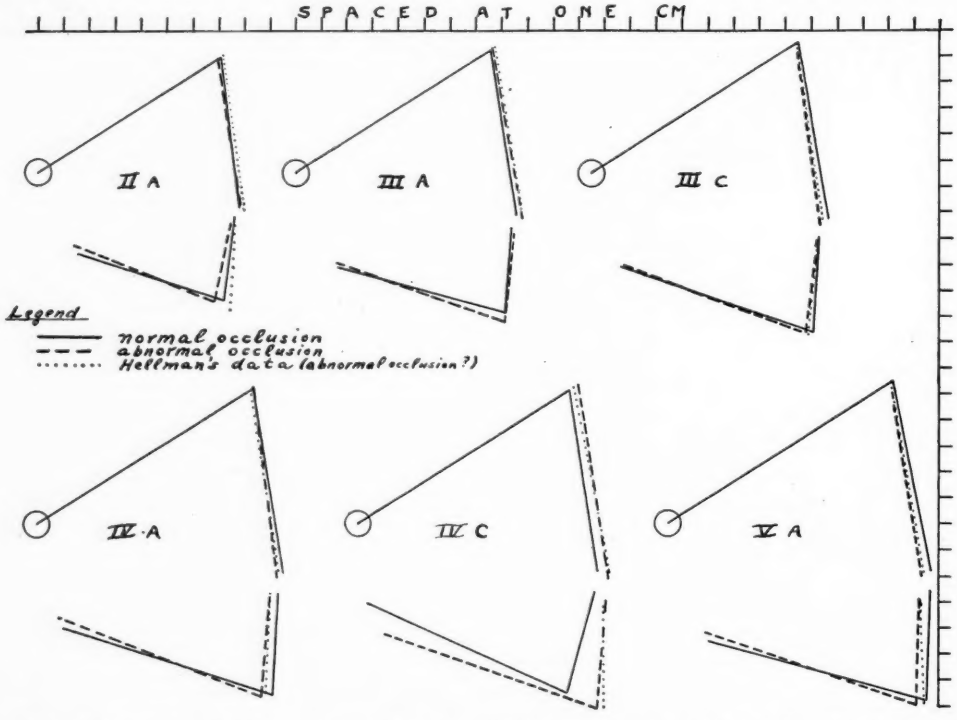


Fig. 2.—Profile of face in normal occlusion compared with facial profile in abnormal occlusion, between dentition stages IIA and VA, respectively.

The most striking observation in this illustration, it seems to us, is a definite tendency for the face of individuals with normal occlusion to grow between stages IIA and VA at a more rapid rate in auricular depth from prosthion down to menton. As a matter of fact, the outline of the face in normal occlusion is greater at every stage but IIIA when depth of face is somewhat greater in abnormal occlusion. At stage IVC the profile representing normal occlusion is based on only 5 cases, and is therefore of questionable validity.

Of interest too is the virtually identical auriculo-nasion depth at all stages except the unreliable IVC in both normal and abnormal occlusion. Depth of face at this level would therefore appear totally unrelated to type of dental occlusion. It is rather the region around the mouth, as would be expected, as

well as the position of the chin which in each instance manifests increasing differentiation in the course of time between individuals with normal dental occlusion and abnormal dental occlusion, respectively.

Hellman's data have been plotted and superimposed on our own figures, as mentioned before. The noted agreement with our profiles representing abnormal occlusion, except at stage IIA, is remarkable. Although Hellman's data comprise both normal and abnormal occlusion combined, it is not unlikely that, since his observations were apparently made largely in private practice, cases with malocclusion predominated in his series.

Mention may be made that detailed comparison of the present material (organized according to chronologic age and regardless of occlusion) with Hellman's data occurs in a previous publication,[12] in which the close agreement of the respective findings is in many instances likewise striking.

Fig. 3 represents total changes between stages IIA and VA, in normal occlusion and malocclusion, respectively. Both in normal occlusion and in abnormal occlusion, total growth has been considerable in length and especially in

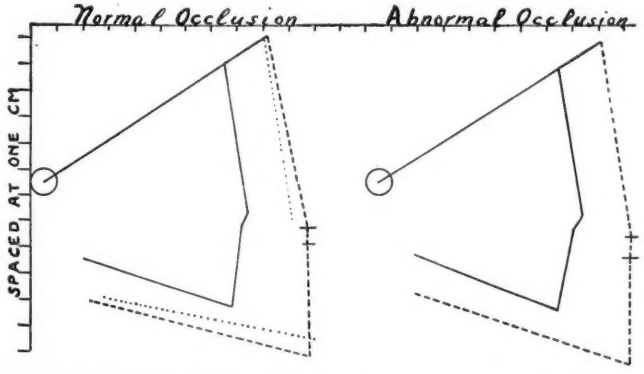


Fig. 3.—Total growth in depth and length of face between dentition stage IIA (solid line) and dentition stage VA (broken line). Effects of senility indicated by dotted line.

depth. Growth in depth, moreover, is evidently greater in the mandibular than in the maxillary region, resulting in a somewhat more prognathous face in the adult.

With regard to occlusion, one can scarcely distinguish the difference in the manner or even extent of growth, when considered in toto, in normal as compared with abnormal occlusion. It must be remembered in this connection, however, that the abnormal category is composed of all types of malocclusion.

There is discernible, too, remarkable stability in the position of the gonion point relative to the ear hole regardless of dental occlusion.

A group of old men, it will be recalled, were also measured. The facial profile of these old men is also plotted (Fig. 3) to show the effects, if any, of senility in the face.

Almost all these men wore artificial dentures which were removed during their examination. Auriculo-infradentale could not be obtained in most cases, necessitating omission of the infradentale-menton projection in the illustration.

The most striking consequence of old age in the face appears to be a shift forward of the whole mandible, suggesting therewith a more obtuse angle of the rami. Such a jutting outward and upward of the chin is indeed not an uncom-

mon observation in old, toothless people. Also evident is a diminution in upper and lower face lengths, undoubtedly the result of loss of teeth. Depth too has been affected, the face has become shrunken, especially around the mouth and probably also due to alveolar absorption after loss of dentition.

### TOTAL INCREMENT

The relative total increment in every character, between stages IIA and VA, should be of interest especially when contrasting normal and abnormal occlusion

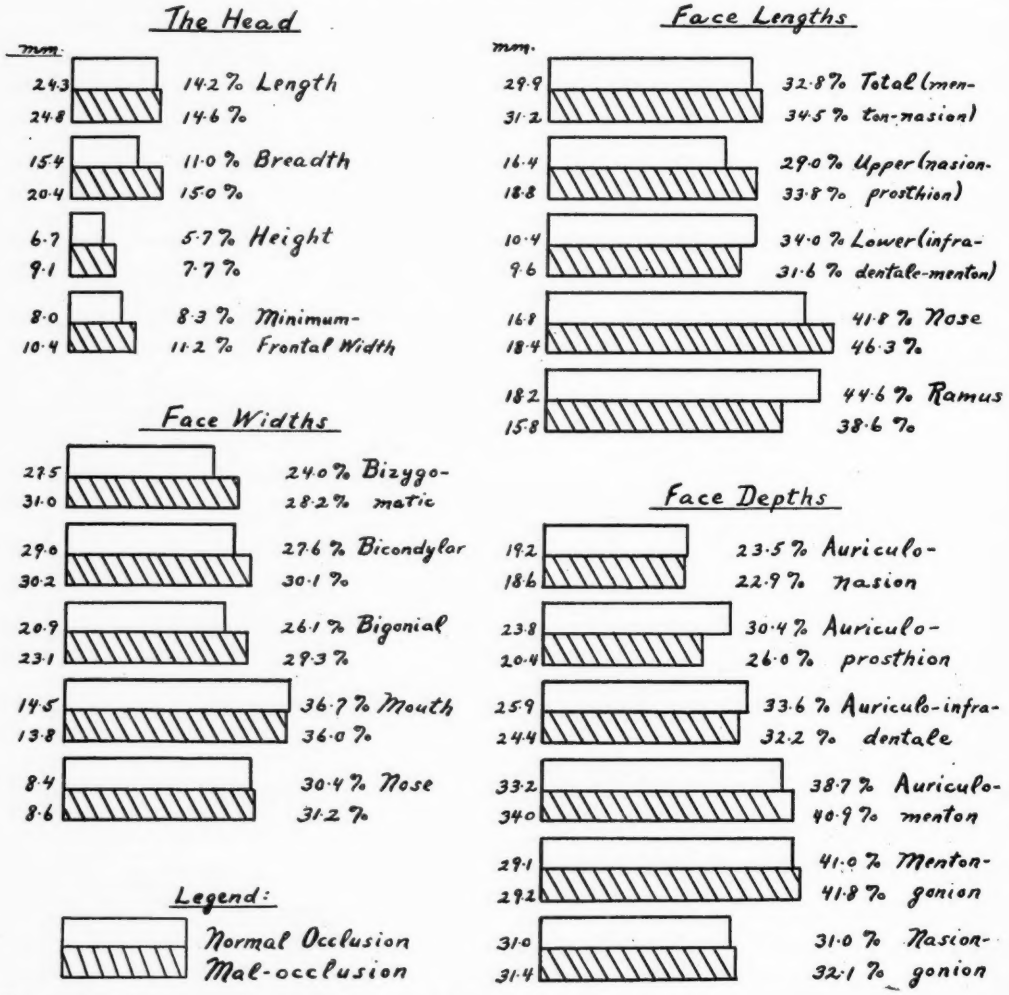


Fig. 4.—Total increments, absolutely and relatively, in the face of subjects with normal occlusion and abnormal occlusion, respectively, between dentition stages IIA and VA.

in this respect. This is done in Fig. 4, in which the bars represent the relative increment indicated to the right, and the figures to the left of the bars indicate the corresponding absolute increments.

The greater amount of growth in width of head and face in abnormal occlusion, it will be recalled, is due to smaller dimensions in this group in early childhood.

Of interest is the tendency for relative total increment in upper face length to be greater in abnormal while increment in the lower face height and height

of ramus are somewhat greater in normal occlusion. Depth of face to the prosthion region manifests an appreciably greater relative increment in normal than in abnormal occlusion.

On the whole, total increment, absolutely and relatively, does not appear to us to be much different in cases with normal occlusion or malocclusion. Marked differences are apparent, however, with respect to character considered. In other words, total relative growth is least in the head, more in width of face, and most in length and depth of face.

Mention may be made that comparison was made between curves of growth, considering cases with normal occlusion and malocclusion, respectively. Very little difference was noted, the conglomerate abnormal curve in many instances actually or almost overlapping that of the normal group. In this connection we may refer the reader to another paper[12] in which rate of growth is treated in detail according to chronologic age. It is here shown that almost all the dimensions of the head and face manifest a peak in rate of growth at three years; following is more or less steady retardation until 13 to 15 years when again there is acceleration of growth, after which retardation is progressive until cessation of increment. The exception to this pattern occurred in widths of the face (except nose) which did not manifest the adolescent spurt.

## VARIABILITY

Variability of the various characters has been measured absolutely by the standard deviation and relatively by the coefficient of variation, presented in Table VII. The number of cases on which these are based is given in the preceding tables. Variability was not computed when too few cases were available.

It will be remembered that the coefficient of variation is a relative measure which makes variability comparable regardless of absolute size; thus, although the absolute variability of nose width is less than that of the bizygomatic width, relatively (to size), variability of nose width is actually much greater than variability of face width.

Comparing relative variability in normal and abnormal occlusion, we note a definite tendency* in early childhood (stage IIA) for widths of the head, and widths and lengths of the face, to be *less* variable in abnormal occlusion, while depths of the face, as well as length of upper face, manifest *greater* variability in abnormal occlusion.

By the time of the full complement of the permanent first molars (stage IIIA), the picture is quite changed. The head (length and width) now manifests greater variability in cases with malocclusion; maximum face width and mandibular width continue to be less variable in malocclusion, but the other face widths and upper and lower face lengths are now more variable in malocclusion. The auricular depths to the mouth region continue to show a higher coefficient in abnormal occlusion, although depth of the mandible (menton-gonion) appears more stable in malocclusion.

At stage IVA, generally the period of adolescence, it is noted that greater variability in abnormal occlusion has become the prevailing tendency in the whole face.

*A difference of 0.5 or more in the coefficient was considered appreciable.

With reference to the question whether variability increases with age (dentition stage here), the evidence on the whole indicates that absolute variability is greater at stage VA than at stage IIA, which would be expected as a

TABLE VII

VARIABILITY OF HEAD AND FACE WITH RESPECT TO NORMAL AND ABNORMAL OCCLUSION

| CHARACTER | OCCLUSION | IIA S. D. MM. | IIA V % | IIIA S. D. MM. | IIIA V % | IIIC S. D. MM. | IIIC V % | IVA S. D. MM. | IVA V % | IVC S. D. MM. | IVC V % | VA S. D. MM. | VA V % |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| *Head:* | | | | | | | | | | | | | |
| Length | Norm. | 6.9 | 4.0 | 5.9 | 3.3 | | | 5.4 | 2.8 | | | | |
| | Ab. | 6.7 | 3.9 | 7.1 | 3.9 | 5.0 | 2.7 | 7.4 | 3.9 | 6.3 | 3.3 | 6.3 | 3.2 |
| Width | Norm. | 5.6 | 4.0 | 4.7 | 3.2 | | | 4.9 | 3.2 | | | | |
| | Ab. | 4.7 | 3.5 | 5.5 | 3.7 | 6.0 | 4.0 | 4.8 | 3.1 | 4.5 | 2.9 | 5.8 | 3.7 |
| Height | Norm. | 4.0 | 3.4 | 4.4 | 3.6 | | | 4.9 | 3.8 | | | | |
| | Ab. | 4.1 | 3.5 | 4.7 | 3.8 | 4.9 | 3.9 | 4.9 | 3.9 | 4.6 | 3.6 | 4.3 | 3.4 |
| Min. Frontal | Norm. | 5.6 | 5.8 | 3.9 | 3.9 | | | 4.1 | 4.0 | | | | |
| | Ab. | 3.7 | 4.0 | 4.3 | 4.3 | 3.2 | 3.1 | 4.2 | 4.1 | 4.4 | 4.2 | 4.9 | 4.8 |
| *Face Widths:* | | | | | | | | | | | | | |
| Bizygomatic | Norm. | 6.7 | 5.8 | 5.8 | 4.7 | | | 4.7 | 3.4 | | | | |
| | Ab. | 5.5 | 5.0 | 5.0 | 4.0 | 4.9 | 3.7 | 5.6 | 4.1 | 4.9 | 3.5 | 5.4 | 3.9 |
| Bicondylar | Norm. | 7.4 | 7.0 | 3.1 | 2.7 | | | 5.2 | 4.0 | | | | |
| | Ab. | 6.4 | 6.3 | 5.4 | 4.7 | 5.6 | 4.7 | 5.9 | 4.6 | 7.6 | 5.7 | 6.2 | 4.7 |
| Bigonial | Norm. | 5.5 | 6.8 | 5.3 | 6.1 | | | 4.8 | 4.9 | | | | |
| | Ab. | 4.3 | 5.4 | 4.9 | 5.5 | 4.1 | 4.4 | 6.1 | 6.2 | 5.0 | 4.9 | 6.5 | 6.3 |
| Nose | Norm. | 2.1 | 7.7 | 1.9 | 6.2 | | | 2.6 | 7.4 | | | | |
| | Ab. | 2.2 | 8.0 | 2.1 | 6.8 | 2.2 | 6.6 | 2.7 | 7.9 | 2.2 | 6.3 | 2.7 | 7.4 |
| Mouth | Norm. | 3.7 | 9.2 | 3.0 | 6.6 | | | 3.2 | 6.4 | | | | |
| | Ab. | 2.8 | 7.4 | 3.4 | 7.5 | 3.6 | 7.6 | 3.1 | 6.3 | 2.7 | 5.3 | 3.3 | 6.3 |
| *Face Lengths:* | | | | | | | | | | | | | |
| Total | Norm. | 5.4 | 5.9 | 5.6 | 5.6 | | | 6.3 | 5.4 | | | | |
| | Ab. | 5.2 | 5.8 | 5.4 | 5.3 | 3.6 | 3.3 | 6.9 | 6.0 | 7.0 | 5.7 | 6.2 | 5.1 |
| Upper | Norm. | 4.2 | 7.4 | 3.2 | 5.1 | | | 4.4 | 6.2 | | | | |
| | Ab. | 4.5 | 8.1 | 3.9 | 6.0 | 2.6 | 3.8 | 4.5 | 6.3 | 4.5 | 6.1 | 4.6 | 6.2 |
| Lower | Norm. | 2.1 | 6.9 | 2.0 | 6.1 | | | 2.9 | 7.5 | | | | |
| | Ab. | 1.9 | 6.3 | 2.6 | 8.1 | 2.5 | 7.1 | 3.1 | 8.3 | 3.1 | 7.8 | 2.7 | 6.7 |
| Nose | Norm. | 3.5 | 8.6 | 3.5 | 7.6 | | | 4.3 | 8.0 | | | | |
| | Ab. | 3.1 | 7.9 | 3.3 | 6.8 | 2.2 | 4.3 | 3.9 | 7.2 | 3.0 | 5.3 | 4.6 | 7.9 |
| Ramus | Norm. | 3.1 | 7.6 | 3.3 | 7.2 | | | 7.0 | 12.1 | | | | |
| | Ab. | 3.7 | 9.0 | 2.9 | 6.3 | 4.4 | 9.1 | 6.2 | 11.1 | 6.5 | 10.9 | 6.1 | 10.8 |
| *Face Depths:* | | | | | | | | | | | | | |
| Auriculo-Nasion | Norm. | 2.9 | 3.5 | 4.0 | 4.6 | | | 3.9 | 4.0 | | | | |
| | Ab. | 4.1 | 5.0 | 4.3 | 4.8 | 3.8 | 4.2 | 4.6 | 4.8 | 3.9 | 4.0 | 3.7 | 3.7 |
| Auriculo-Prosthion | Norm. | 4.1 | 5.2 | 3.7 | 4.3 | | | 4.5 | 4.7 | | | | |
| | Ab. | 4.9 | 6.2 | 4.6 | 5.3 | 4.3 | 4.7 | 5.4 | 5.8 | 4.8 | 4.9 | 5.1 | 5.2 |
| Auriculo-Infradentale | Norm. | 4.2 | 5.5 | 3.8 | 4.5 | | | 4.9 | 5.1 | | | | |
| | Ab. | 5.1 | 6.7 | 4.4 | 5.1 | 3.7 | 4.1 | 6.1 | 6.5 | 5.2 | 5.2 | 6.1 | 6.0 |
| Auriculo-Menton | Norm. | 6.1 | 7.1 | 5.5 | 5.8 | | | 6.5 | 5.9 | | | | |
| | Ab. | 5.7 | 6.8 | 4.8 | 5.0 | 5.0 | 4.9 | 7.0 | 6.5 | 5.5 | 4.8 | 7.0 | 6.0 |
| Menton-Gonion | Norm. | 4.2 | 5.9 | 5.1 | 6.5 | | | 5.3 | 5.4 | | | | |
| | Ab. | 4.3 | 6.1 | 4.2 | 5.2 | 5.8 | 6.6 | 5.1 | 5.3 | 4.1 | 4.1 | 5.2 | 5.2 |
| Nasion-Gonion | Norm. | 4.9 | 4.9 | 6.4 | 5.8 | | | 5.8 | 4.6 | | | | |
| | Ab. | 4.7 | 4.8 | 5.1 | 4.6 | 4.2 | 3.6 | 6.2 | 5.0 | 5.0 | 3.8 | 4.5 | 3.5 |

result of mere increase in size with growth. Relatively, however, variability in most instances actually diminishes with age, a phenomenon which holds true for growth in general.

Considering degree of variability with reference to plane of face, in general, variability is greatest in height of face, less in width, and least in depth. The head, however, manifests even less variability than depth of face.

VARIOUS TYPES OF MALOCCLUSION

One of the striking facts revealed in the preceding was the close similarity in many of the average dimensions in subjects with normal occlusion and those with abnormal occlusion. This similarity was candidly looked at with suspicion, and for a simple and often overlooked reason. It is a matter of common knowledge that there are many and strikingly diverse kinds of malocclusion. Not-

TABLE VIII

AVERAGE DIMENSIONS (MM.) IN NORMAL OCCLUSION AND GOOD BITE AND MEAN DEVIATION THEREFROM IN THE SEVERAL TYPES OF BITE

| BITE | STAGE IIA | | | | STAGE IIIA | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | NORM. | ABNORMAL | | | NORMAL | | ABNORMAL | | | |
| | GOOD (37) | EX-CESS (33) | CLASS II (27) | OPEN (16) | GOOD (10) | POOR (12) | GOOD (22) | EXCESS (63) | ED.-ED. (7) | OPEN (4) |
| Age (yr.) | 4.45 | 3.85 | 4.58 | 3.34 | 7.88 | 8.32 | 8.73 | 9.04 | 8.2 | 7.7 |
| *Head:* | | | | | | | | | | |
| Length | 170.8* | -0.6 | +7.4 | -3.0 | 179.0 | -1.0 | -3.0 | +1.1 | +5.0 | -3.0 |
| Width | 140.6 | -5.2 | +0.1 | -6.6 | 146.0 | 0.0 | 0.0 | +0.8 | +1.0 | -2.0 |
| Height | 118.3 | -1.3 | +6.6 | -1.3 | 123.0 | -2.0 | 0.0 | -0.5 | 0.0 | 0.0 |
| Minim. Frontal | 96.0 | -3.4 | +0.3 | -4.1 | 101.0 | -1.0 | 0.0 | -0.9 | 0.0 | -3.0 |
| *Face Widths:* | | | | | | | | | | |
| Bizygomatic | 114.5 | -4.9 | -0.5 | -6.5 | 123.0 | +2.0 | +1.0 | +2.4 | +3.0 | -1.0 |
| Bicondylar | 105.0 | -4.9 | -0.5 | -7.0 | 114.0 | +1.0 | -2.0 | -0.2 | -1.0 | -2.0 |
| Bigonial | 80.1 | -1.8 | +0.1 | -2.1 | 85.0 | +4.0 | +2.0 | +4.4 | +3.0 | +3.0 |
| Nose | 27.6 | 0.0 | -0.6 | -0.6 | 30.0 | 0.0 | 0.0 | +0.7 | -1.0 | +1.0 |
| Mouth | 39.5 | -1.2 | -1.0 | -1.6 | 45.0 | 0.0 | -1.0 | +0.2 | -1.0 | +1.0 |
| *Face Lengths:* | | | | | | | | | | |
| Total | 91.1 | -1.3 | +1.8 | -1.6 | 97.0 | +4.0 | +6.0 | +6.2 | +7.0 | +7.0 |
| Upper | 56.6 | -0.4 | +2.3 | -3.6 | 61.0 | +3.0 | +2.0 | +4.6 | +4.0 | +4.0 |
| Lower | 30.6 | -0.4 | +0.8 | -0.6 | 32.0 | 0.0 | 0.0 | +0.9 | 0.0 | 0.0 |
| Nose | 40.2 | -0.3 | -0.5 | -2.2 | 46.0 | +1.0 | +1.0 | +2.3 | +1.0 | +2.0 |
| Ramus | 40.8 | +0.2 | +1.0 | 0.0 | 45.0 | +1.0 | +1.0 | +0.9 | -2.0 | +1.0 |
| *Face Depths:* | | | | | | | | | | |
| Auric.-Nasion | 81.8 | -0.9 | +2.9 | -1.0 | 86.0 | +1.0 | +3.0 | +2.3 | +3.0 | +2.0 |
| Auric.-Prosth. | 78.2* | -0.1 | +1.3 | +1.3 | 86.0 | 0.0 | -1.0 | +1.3 | -2.0 | 0.0 |
| Auric.-Infrad. | 77.1† | -2.6 | -0.9 | +0.7 | 86.0 | -2.0 | -1.0 | +0.3 | +1.0 | +2.0 |
| Auric.-Menton | 85.8* | -3.6 | -0.7 | -2.2 | 95.0 | +1.0 | +3.0 | +2.7 | +2.0 | +2.0 |
| Menton-Gonion | 70.9 | -1.5 | +5.0 | -0.4 | 78.0 | +1.0 | +2.0 | +2.9 | +1.0 | 0.0 |
| Nasion-Gonion | 100.0 | -1.9 | +4.5 | -4.0 | 108.0 | +2.0 | +1.0 | +3.0 | +1.0 | +2.0 |

NOTE.—Figures in parentheses refer to number of cases.

The minus or plus signs indicate the divergence from the averages in normal occlusion. Under normal, "good" refers to a good normal; "poor," a poor normal (slight rotation of a tooth or slight break in surface-to-surface contact of incisors). Under abnormal, "good" refers to cases which have lost a tooth, the occlusion otherwise remaining undisturbed; "excess," too much horizontal and vertical overbite; "open," open-bite of the incisors; "ed.-ed.," an edge-to-edge contact of the incisors with the teeth in centric occlusion; "ret." retrusive maxillary incisors; i.e., mandibular incisors in front of maxillary with teeth in centric occlusion. All these kinds of malocclusion are in the Angle Class I category; Class II and Class III, respectively, refer to Angle's classification as such.

*Based on 36 cases.

†Based on 33 cases.

withstanding this fact, and its obvious implication that the mode of development may differ in the different kinds of malocclusion, normal occlusion has been and is usually compared with a single, conglomerate group including all the various forms of malocclusion. Such a combined abnormal occlusion group may easily obscure or even neutralize important differences in absolute dimensions and rates of growth. A face with a jutting or relatively overdeveloped

TABLE IX

AVERAGE DIMENSIONS (MM.) IN NORMAL OCCLUSION AND GOOD BITE AND MEAN DEVIATION THEREFROM IN THE SEVERAL TYPES OF BITE

| BITE | STAGE IIIC | | | | | STAGE IVA | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | NORM. | ABNORMAL | | | | NORMAL | | ABNORMAL | | | | | |
| | GOOD (4) | GOOD (7) | EXCESS (9) | CLASS II (4) | RET. (2) | GOOD (34) | POOR (10) | GOOD (56) | EXCESS (36) | CLASS II (14) | CLASS III (2) | RET. (2) | OPEN (6) |
| Age (yr.) | 13.06 | 11.96 | 13.42 | 13.06 | 11.71 | 16.14 | 13.88 | 15.42 | 15.20 | 14.66 | 17.50 | 16.90 | 15.04 |
| *Head:* | | | | | | | | | | | | | |
| Length | 182.0 | +3.6 | +1.2 | +8.3 | +5.0 | 191.6 | -4.6 | -1.3 | -1.7 | -2.1 | -1.6 | -0.6 | +1.2 |
| Width | 150.0 | +0.4 | -3.2 | +1.0 | +4.0 | 154.8 | +0.2 | -0.5 | -0.9 | -4.2 | +3.7 | +0.2 | 0.0 |
| Height | 123.5 | +2.9 | +1.7 | +3.0 | +4.0 | 127.2 | -1.2 | -0.5 | -1.5 | -1.9 | +2.3 | -2.2 | +0.4 |
| Minim. Front. | 99.0 | +4.0 | +1.8 | +6.0 | -2.0 | 103.4 | -0.4 | -0.5 | +0.5 | +0.3 | +0.1 | -2.9 | +1.7 |
| *Face Widths:* | | | | | | | | | | | | | |
| Bizygomatic | 130.0 | -0.3 | -0.3 | 3.3 | -0.5 | 137.9 | -3.9 | -1.9 | -0.6 | -3.5 | +5.6 | -0.4 | -1.6 |
| Bicondylar | 119.0 | -1.7 | +0.4 | +5.8 | +1.5 | 128.6 | -3.6 | -2.7 | -0.5 | -3.7 | +1.4 | -0.6 | -0.3 |
| Bigonial | 92.0 | -0.4 | +1.8 | +4.5 | +0.5 | 99.9 | -2.9 | -1.4 | -1.4 | -3.3 | -1.4 | -5.4 | -1.2 |
| Nose | 33.5 | -0.8 | -2.2 | +0.5 | -1.0 | 35.4 | -1.4 | -1.0 | -0.4 | -1.3 | -1.4 | -2.9 | -1.9 |
| Mouth | 47.0 | +1.6 | +0.2 | +0.3 | -2.0 | 49.8 | +0.2 | -0.4 | +0.2 | -1.0 | -2.3 | -1.8 | -1.5 |
| *Face Lengths:* | | | | | | | | | | | | | |
| Total | 110.0 | -2.3 | -0.6 | +2.0 | -1.5 | 117.9 | -8.9 | -0.8 | -5.4 | -1.3 | +3.6 | -2.9 | -1.1 |
| Upper | 68.0 | 0.0 | +1.1 | +1.3 | +2.8 | 71.5 | -4.5 | +0.5 | -1.0 | +2.5 | +1.5 | -2.0 | -1.2 |
| Lower | 36.0 | -2.1 | -0.9 | +1.0 | -3.5 | 38.7 | -2.7 | -0.7 | -2.3 | -0.3 | +2.3 | +0.3 | -2.0 |
| Nose | 51.0 | +0.4 | -0.3 | -2.5 | -1.0 | 54.4 | -3.4 | -0.3 | -1.5 | -0.6 | +1.6 | -1.4 | -3.4 |
| Ramus | 50.7 | -3.6 | -2.9 | +3.8 | -3.4 | 58.7 | -4.7 | -2.3 | -4.7 | -3.3 | 1.8 | -10.7 | -3.9 |
| *Face Depths:* | | | | | | | | | | | | | |
| Auric.-Nasion | 92.5 | -0.1 | -1.4 | +1.0 | -2.5 | 97.8 | -4.8 | -0.9 | -1.5 | +6.3 | -1.8 | -5.8 | -0.6 |
| Auric.-Prosth. | 91.5 | -2.4 | +0.3 | +2.0 | -4.5 | 96.3 | -2.3 | -2.5 | -2.5 | -0.6 | -7.8 | -7.3 | -4.3 |
| Auric.-Infrad. | 89.7 | +0.3 | +0.7 | +1.1 | -1.7 | 96.5 | -3.5 | -2.5 | -4.1 | -3.6 | -0.5 | -3.0 | -3.5 |
| Auric.-Menton | 103.7 | -2.4 | -2.1 | +2.3 | -5.7 | 111.6 | -3.6 | -2.7 | -3.9 | -4.4 | +0.9 | -4.6 | -2.0 |
| Menton-Gonion | 89.7 | -4.4 | -4.0 | +6.1 | -3.4 | 97.7 | -2.7 | -1.1 | -2.6 | -3.3 | +4.3 | -5.2 | -0.7 |
| Nasion-Gonion | 118.0 | -2.1 | -1.9 | +2.8 | -4.0 | 127.6 | -7.6 | -2.9 | -4.2 | -1.9 | -1.6 | -6.6 | -2.8 |

Figures in parentheses refer to number of cases.

*upper* jaw, for example, certainly differs from a face with a jutting or relatively overdeveloped *lower* jaw. Combine these two forms, however, and a normal profile is likely to result.

We have taken cognizance of this problem by splitting abnormal occlusion into several principal types and analyzing the development of the head and face with regard to each of these subdivisions. One great difficulty in such a refinement of method is that the series in each subdivision becomes small and the reliability of the averages is consequently weakened. This difficulty can be

TABLE X

AVERAGE DIMENSIONS (MM.) IN NORMAL OCCLUSION AND GOOD BITE AND MEAN DEVIATION THEREFROM IN THE SEVERAL TYPES OF BITE

| BITE | STAGE IVC | | | | STAGE VA | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | NORM. | ABNORMAL | | | NORM. | ABNORMAL | | | | | |
| | GOOD | GOOD | EXCESS | RET. | GOOD | GOOD | EX-CESS | CLASS II | CLASS III | RET. | OPEN |
| | (5) | (15) | (8) | (3) | (13) | (10) | (12) | (4) | (2) | (2) | (3) |
| Age (yr.) | 19.25 | 18.45 | 18.82 | 18.47 | 20.17 | 19.4 | 19.78 | 19.69 | 19.75 | 19.00 | 19.97 |
| *Head:* | | | | | | | | | | | |
| Length | 187.0 | +7.1 | +7.0 | +11.3 | 194.0 | +3.4 | -0.8 | 0.0 | +3.0 | -3.0 | +5.0 |
| Width | 154.0 | +3.0 | +2.1 | +1.7 | 156.0 | -0.1 | -0.2 | +1.7 | +2.0 | +1.5 | +0.6 |
| Height | 126.0 | +2.8 | +0.5 | +4.7 | 125.0 | +2.2 | +1.2 | +1.0 | +4.0 | +2.0 | +1.3 |
| Minim. Front. | 100.0 | +3.8 | +5.1 | +4.0 | 104.0 | -1.7 | -0.7 | -1.3 | +2.0 | -3.0 | -0.7 |
| *Face Widths:* | | | | | | | | | | | |
| Bizygomatic | 139.0 | +2.8 | +0.6 | +2.0 | 142.0 | -2.1 | -1.5 | 0.0 | 0.0 | -2.0 | -0.7 |
| Bicondylar | 132.0 | +1.0 | -2.5 | -1.7 | 134.0 | -3.7 | -4.7 | -1.5 | -1.0 | -2.5 | -2.0 |
| Bigonial | 103.0 | -0.3 | -0.7 | -1.0 | 101.0 | +1.4 | +0.8 | -1.0 | +4.0 | +1.5 | +2.6 |
| Nose | 35.0 | +0.7 | -1.0 | +0.3 | 36.0 | +0.6 | -0.5 | -0.3 | +2.5 | -1.0 | +1.0 |
| Mouth | 53.0 | -2.8 | -1.6 | -0.3 | 54.0 | -1.3 | -2.2 | -4.3 | +2.0 | -1.0 | -2.7 |
| *Face Lengths:* | | | | | | | | | | | |
| Total | 114.0 | +7.4 | +5.3 | +10.6 | 121.0 | +1.6 | 0.0 | -1.5 | +7.5 | +5.0 | +1.3 |
| Upper | 69.0 | +4.6 | +3.9 | +4.3 | 73.0 | +2.3 | +2.2 | 0.0 | +1.5 | 0.0 | +1.7 |
| Lower | 39.0 | +1.5 | -0.5 | +2.3 | 41.0 | -0.9 | -1.6 | -1.3 | 0.0 | +1.5 | -0.4 |
| Nose | 53.0 | +2.8 | +2.8 | +1.3 | 57.0 | +0.9 | +2.3 | -1.2 | +6.5 | -0.5 | +0.6 |
| Ramus | 55.0 | +5.2 | +4.8 | +0.3 | 59.0 | -1.0 | -4.9 | -1.7 | -4.0 | -2.5 | +1.6 |
| *Face Depths:* | | | | | | | | | | | |
| Auric.-Nasion | 95.0 | +3.5 | +3.9 | +5.7 | 101.0 | -0.5 | -1.2 | -3.5 | +0.5 | -3.0 | +0.3 |
| Auric.-Prosth. | 93.0 | +3.9 | +6.5 | +5.0 | 102.0 | -2.4 | -3.7 | -2.5 | +2.0 | -8.0 | -2.4 |
| Auric.-Infrad. | 94.0 | +3.9 | +6.1 | +8.0 | 103.0 | -2.4 | -4.7 | -2.8 | +8.0 | -5.0 | 0.0 |
| Auric.-Menton | 112.0 | +0.9 | +5.0 | +5.3 | 119.0 | -2.0 | -2.8 | -2.7 | +13.5 | -2.5 | -3.0 |
| Menton-Gonion | 98.0 | +2.0 | +0.3 | +4.3 | 99.0 | +0.6 | -2.7 | +0.5 | +4.5 | +1.5 | +2.0 |
| Nasion-Gonion | 123.0 | +6.4 | +6.9 | +6.3 | 131.0 | -0.6 | -1.2 | -2.0 | 0.0 | -6.5 | -2.7 |

Figures in parentheses refer to number of cases.

overcome, of course, by observing sufficiently large numbers of individuals with each type of malocclusion; this is a huge task, it is true, yet quite essential. In the present instance, regrettably enough, the distribution of cases in the various subdivisions of abnormal occlusion is uneven; in some instances the series are quite respectable, in others they are not. Nevertheless, inasmuch as the manner of presentation may in itself be of interest, and the results seem to us of considerable significance, the whole data are given in some detail in Tables VIII, IX, and X.

It may be well first to clarify the meaning of the subdivisions used. Normal occlusion was broken up into two classes: cases with "good" normal occlusion,

and cases with "poor" normal occlusion, respectively. An example of the latter would be slight rotation or crowding of the incisors, or a slight break in the surface-to-surface contact of the central incisors.

Abnormal occlusion was subdivided into Angle's Classes II and III, respectively; all the other subdivisions refer to Angle's Class I, which was split up with regard to "bite" or kind of interrelationship of the maxillary and mandibular teeth *in local sections* of the dental arch. The "good" division in the latter refers to cases, for example, wherein teeth have been lost but aside from this there has been no serious disturbance in the occlusion. The "excess" category means too much horizontal and vertical overbite of the maxillary anterior teeth. A previous paper[17] has demonstrated that excess vertical overbite is usually coincident with excess horizontal overbite, and hence their combination. "Open-bite" refers to the incisor region; "ed.-ed." means an edge-to-edge contact of the central incisors when normally closed; "ret." refers to a retrusive bite of the maxillary incisors, that is, the mandibular incisors are in front of the maxillary incisors when the teeth are in centric occlusion.

Let us now examine the tabular material. The average dimensions are given only for good normal occlusion; under each type of abnormal occlusion is the divergence, plus or minus, from this average in normal good occlusion. Thus, under the excess column at stage IIA (Table VIII), –0.6 mm. in length of head simply means that the average length of head in subjects with excessive dental overbite is 0.6 mm. *less* than in subjects with good normal occlusion; a plus sign would mean that the particular average is *greater* to the extent indicated, than the corresponding dimension in normal occlusion.

(a) *The Head.*—The results indicated in Table VIII are striking. Considering the head, we note first that the groups with the various types of malocclusion are at times markedly divergent, one type from another as well as each from the normal occlusion group. This is true, moreover, not only in the width of head but also in length and in height. Differences to the extent of 7 and even 11 mm. are noted between comparable averages at the same dentition stage.

Second is a tendency for the differences between each of the several occlusion types to diminish in the latter dentition stages, although the trend is not consistent in that appreciable divergence from normal occlusion occurs at all stages.

Third, the divergence from normal occlusion tends to be in one direction, plus or minus, especially in some types of malocclusion, as Class III and retrusive bite, for example.

The reason for these results is difficult to state. There is a complicating factor of averages based on very small series, as in Class III type of malocclusion. To be sure, this form of malocclusion is relatively rare in the general population, and employment of a few cases only is perhaps justifiable in this instance for the determination of conditions in the face, and especially development around the dental regions. But the head proper of these cases may not be at all related to the dental malocclusion; hence averages based on small numbers so far as the head dimensions are concerned may not be indicative of prevailing conditions.

Too, there is the question of age. It will be noted that the average age of the individuals in each of the occlusion subdivisions varies more or less even though in the same dentition stage. At dentition stage IIA, for example, the average of the excessive bite group is appreciably less than that of the normal good group; moreover the dimensions in the former seem correspondingly less than the dimensions in the latter. Yet at this same dentition stage (IIA) Class II malocclusion is about the same average age as is the normal occlusion group, with dimensions of the former, however, much greater than those of the latter.

In short, the significance of these albeit appreciable differences in the head dimensions is not clear, and again we must say that additional work along the same or similar lines is necessary before a definite conclusion can be drawn on the relationship of the various types of malocclusion with growth of the head.

(b) *Face Widths.*—Turning to width of face, we find here, too, substantial differences between each of the malocclusion types and these from normal occlusion, in both the early and the later dentition stages. The direction of divergence (plus or minus) is not the same in all the facial widths at any single stage, nor at all stages in any single width diameter.

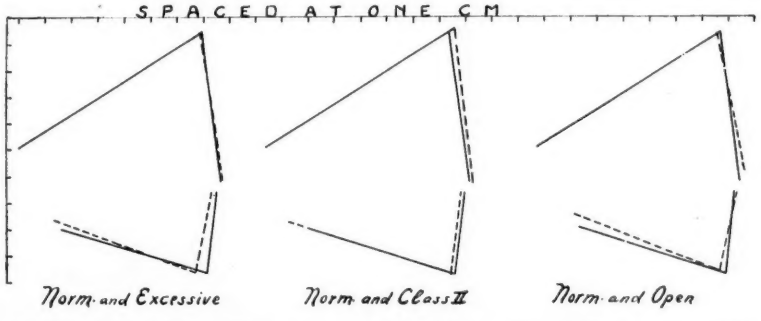


Fig. 5.—Comparison of facial profile in normal occlusion with profile in the various types of malocclusion at dentition stage IIA (solid line, Normal; broken line, Abnormal).

(c) *Face Lengths and Depths.*—Face lengths and depths will be considered together, as graphically represented in facial profiles. At each dentition stage the profile of the face in good normal occlusion is compared with each of the facial profiles representing the several other types of occlusion.

*Dentition Stage IIA.*—Fig. 5 illustrates conditions at this time. We note in excessive overbite (horizontal and vertical, Angle's Class I) little or no difference from the normal profile in the upper part of the face; the lower portion, however, comprising the whole mandible, is shifted backward relative to conditions in normal occlusion.

Comparison of cases with normal occlusion and Class II malocclusion is quite interesting.* The upper face in the latter is deeper and slightly longer; apparent lack of growth in auricular depth in the mandible seems actually a mere shift back of the mandible, perhaps growth backward instead of forward.

Open-bite compared with normal is also illuminating, showing that it is a shortening in upper face length which marks this type of malocclusion. Thumb-sucking, the habit which is primarily the cause of open-bite at this time, ap-

*It should be remembered that excessive overbite is also often characteristic of Class II malocclusion, in addition to the mesiodistal disturbance in the cheek teeth of the latter.

parently affects only the maxillary teeth and alveolar process. Of interest, too, is the greater alveolar prognathism in the open-bite profile, and here, too, there is a slight shifting backward of the body of the mandible.

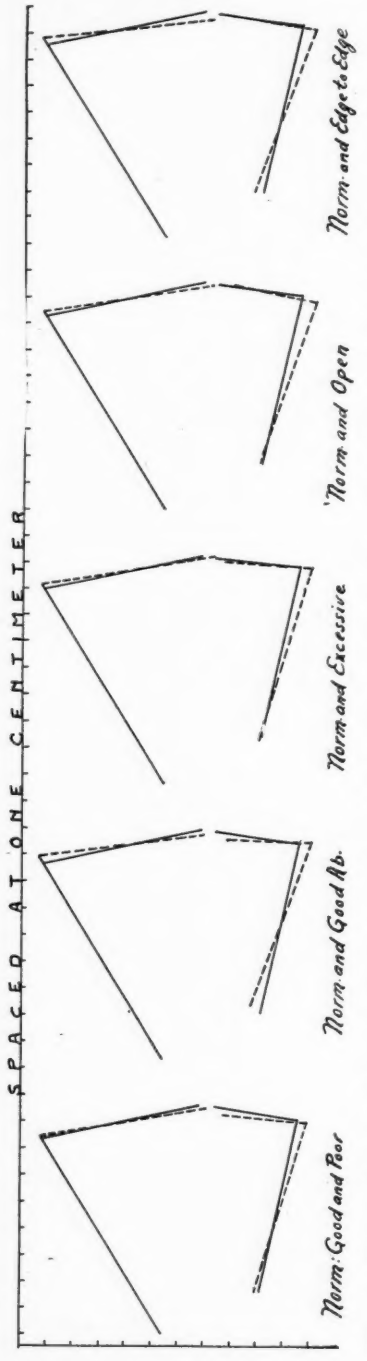


Fig. 6.—Comparison of facial profile in normal occlusion with profile in the various types of malocclusion at dentition stage IIIA (solid line, Normal; broken line, Abnormal).

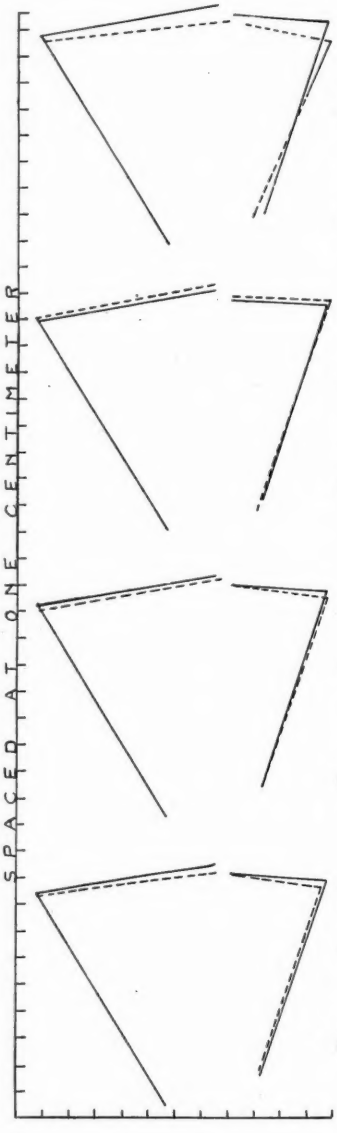


Fig. 7.—Comparison of facial profile in normal occlusion with profile in the various types of malocclusion at dentition stage IIIC (solid line, Normal; broken line, Abnormal).

*Dentition Stage IIIA.*—(Fig. 6.) At this stage, with all the permanent first molars erupted, our material affords us the interesting comparison of conditions in good normal occlusion and poor normal occlusion, as well as comparison of good normal with good abnormal. It is at once noted that the poor

normal profile manifests a longer face than in good normal occlusion, apparently as a result of greater upper facial length which in turn has evidently pushed downward the whole symphysis region of the mandible. Lack of growth in depth in the infradentale region also is apparent in "poor" normal occlusion. The good *abnormal* profile is not dissimilar to the poor *normal,* as was expected.

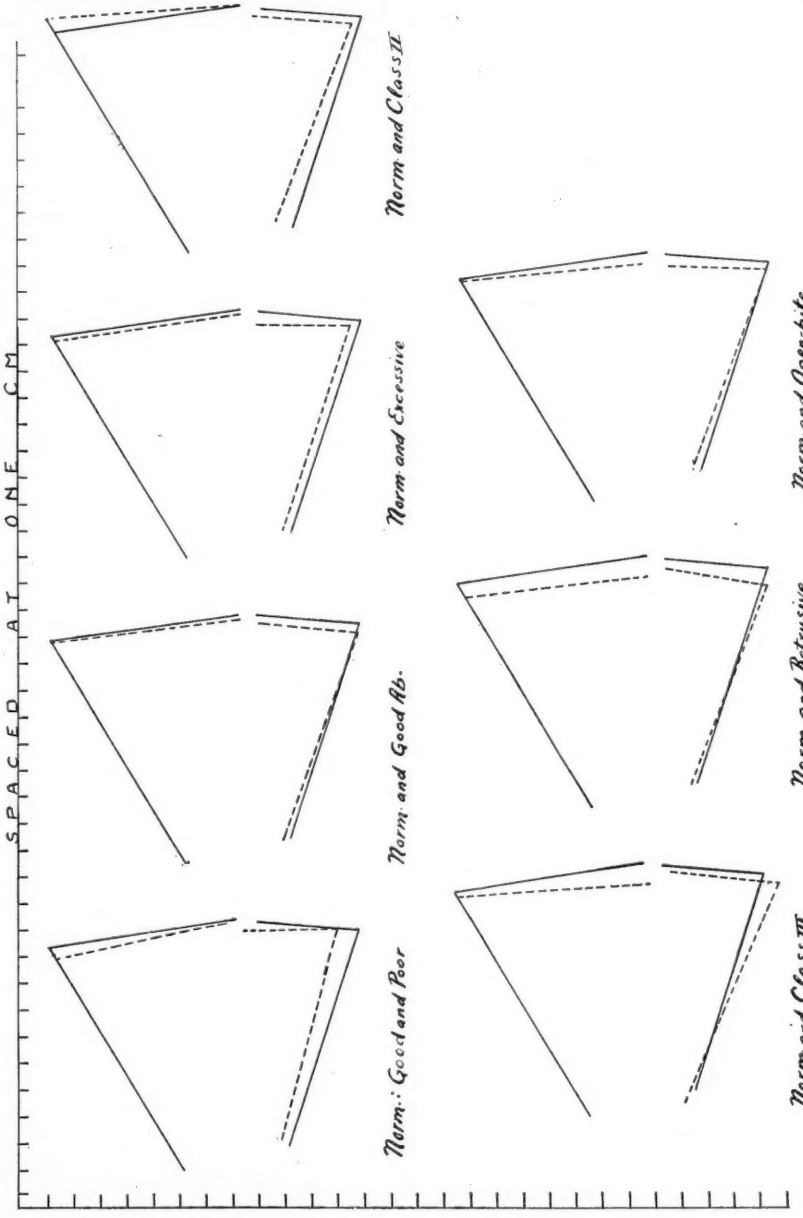


Fig. 8.—Comparison of facial profile in normal occlusion with profile in the various types of malocclusion at dentition stage IVA (solid line, Normal; broken line, Abnormal).

Relative to the normal, the good abnormal is shorter in depth in both the prosthion and the infradentale regions, especially in the latter. Total face length is also evidently greater in the abnormal group, as it is, in fact, in all the other types of abnormal occlusion at this dentition stage. Depth of the upper part of the face (auriculo-nasion) is likewise somewhat more in this and all the other malocclusion groups.

The excessive overbite face manifests both similar and different tendencies when compared with the normal, in this dentition stage as in the previous (IIA). We note that the upper face length has become much longer relative to this dimension in normal occlusion, in consequence of which there has evidently been a pushing downward of the whole chin region. This time, however, the relative forward position of the infradentale and menton is virtually the same as in normal occlusion in contrast to their appreciably anterior position (relative to the normal profile) at dentition stage IIA.

The picture of the face with open-bite of the anterior teeth, in comparison with the profile in normal occlusion, is remarkably different from that observed at stage IIA. Upper face length is now greater in open-bite, with the infradentale point much lower as a result of the lower position of the entire chin region, all in direct contrast to conditions at stage IIA.

Regarding the profile of the face of subjects with an edge-to-edge bite of the incisors, we note that a lag in growth outward in the maxillary alveolar region is apparently responsible for this type of malocclusion.

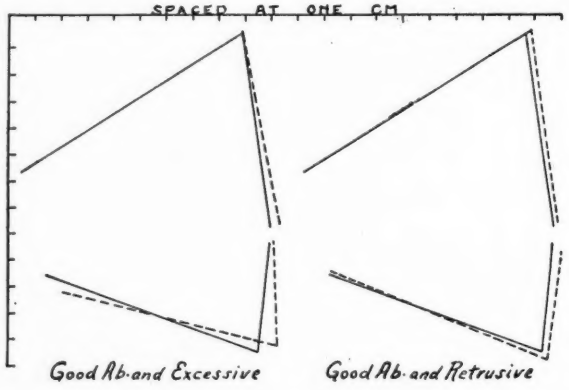


Fig. 9.—Comparison of facial profile in normal occlusion with profile in the various types of malocclusion at dentition stage IVC (solid line, Normal; broken line, Abnormal).

*Dentition Stage IIIC.*—(Fig. 7.) The number of cases representing this stage in any type of occlusion is small; yet the comparative figures are shown as tentative indications of the conditions existing.

Of particular interest are the Class II and retrusive pictures. In the former we have again, as at stage IIA, greater growth in depth relative to dimensions in normal occlusion, and in the present dentition stage this is so in the lower as well as in the upper face. In retrusive bite, which simulates in symptomatology the Class III type of malocclusion, deficiency of growth in depth of face is striking; this lag evidently occurs more severely in the prosthion than in the infradentale region. Length of face is apparently not affected.

*Dentition Stage IVA.*—(Fig. 8.) The averages at this stage are more representative so far as numbers are concerned except perhaps the poor normal group whose profile is, indeed, widely divergent from that of the good normal. It is of much interest and probable significance that by the time of adolescence or approaching adolescence (second permanent molars), the face at all levels is more or less shorter in depth in almost every type of malocclusion as compared

with conditions in normal occlusion. This is especially true in the retrusive bite profile, as was also noted in the preceding dentition stage.

Comparison of the Class III and retrusive profiles, in relation to normal occlusion, is of peculiar interest in view of the similar symptomatology usually manifested by each of these forms of malocclusion. Lack of development in Class III is obviously greater in the maxillary alveolar region than in the mandibular at this time. Now, although a like situation is evident in retrusive bite, the difference is here far less distinct. As a result of this, the interrelationship of the teeth anteriorly, even, is much more adversely affected in Class III malocclusion. The face in Class III malocclusion, moreover, appears longer in total length and in depth of the body of the mandible.

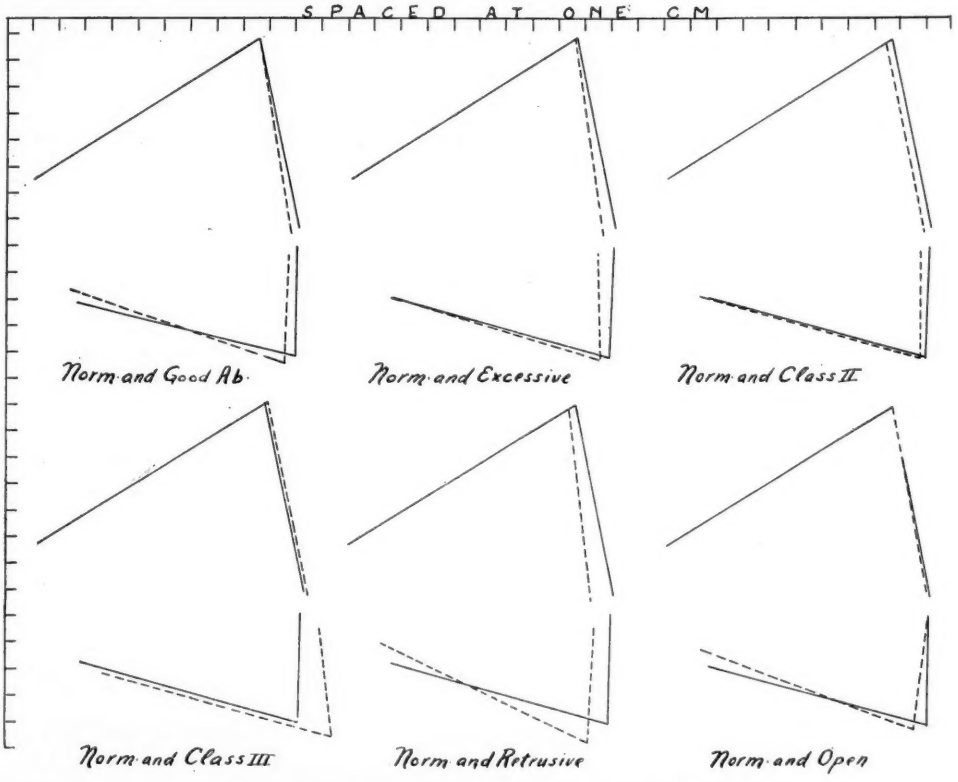


Fig. 10.—Comparison of facial profile in normal occlusion with profile in the various types of malocclusion at dentition stage VA (solid line, Normal; broken line, Abnormal).

The exception to subnormal depth of face at all levels is Class II malocclusion. Conditions here, in fact, are essentially similar to the relative relationship of normal occlusion and Class II at dentition stage IIA. The auriculonasion depth manifests appreciably more outward growth than in normal occlusion. On the other hand, the mandible appears quite less developed than in normal occlusion.

Excessive overbite is likewise at this time, as indeed was the case at stage IIA, distinguished by lack of mandibular development, especially in depth.

Open-bite manifests slight shortness in length of face in addition to shortness in depth, in both the maxillary and the mandibular alveolar regions.

*Dentition Stage IVC.*—(Fig. 9.) The number of subjects with normal occlusion was so negligible that a profile was not made for this group; instead, good bite of abnormal occlusion was taken as the profile of comparison.

The rather appreciably greater depth of face noted in the excessive profile is contrary to the usual condition observed in the preceding dentition stages; namely, approximately the same or less development as normal, especially around the mouth region. In fact, even the series representing excessive overbite is at this stage far too small for reliable deductions, being given merely as a matter of record.

The profile of retrusive bite is likewise unexpectedly greater in depth than in good abnormal.

*Dentition Stage VA.*—(Fig. 10.) Several remarkable tendencies are noted here, the adult stage. The Class II profile is now *less* in depth, at all levels of the face, than in normal occlusion. Excessive overbite also manifests lack of development in depth, a general tendency already commented upon in the preceding. It is of interest to note the similarity of facial profiles in subjects with excessive overbite (Angle's Class I) and Angle's Class II malocclusion, the latter, in fact, usually also manifesting pronounced protrusion in the anterior portion of the maxillary arch.

Retrusive bite is still short in depth, but, remarkably enough, the face in Class III malocclusion is now greater in depth than normal. There has been, moreover, a striking forward shift of the whole mandible relative both to position in normal occlusion and to the maxilla in Class III malocclusion itself.

Open-bite malocclusion seems now, as at stage IVA, a matter of insufficiency in the anterior alveolar region in both the maxilla and the mandible, in contrast with the deficiency in the prosthion region only at stage IIA.

A few words may not be amiss regarding some of the figures based on small series. It is a matter of fact[17] that Class III malocclusion, or retrusive bite in Angle's Class I category, is in each instance comparatively rare in the general population. Edge-to-edge bite of the incisors, too, is quite infrequent except perhaps in middle age or old age when worn teeth may induce such a condition. Open-bite of the incisors, also, is prevalent only in infancy or early childhood, primarily as a result of thumb-sucking. Even Angle's Class II type of malocclusion is apparently not common at certain ages. Hence, it would be and is difficult for any single investigator to get many cases with these forms of malocclusion even in fairly large samples of the general population. Indeed, under the circumstances even a comparatively few cases might well be a fair percentage of the total incidence and the data pertaining to these few consequently be indicative of the prevailing tendencies.

However this may be, we must confess that larger representations in each of the above mentioned forms of malocclusion would have been much more desirable, not merely as assurance of greater reliability in average values, but also as a source for determining extent of variability, something which was scarcely possible with the material available. Indeed, Hellman[9] has admirably pointed out the wide variability in the face even in cases with a special type of malocclusion like the Class III.

It should be mentioned that Hellman[11] found open-bite "in constant relationship with an absolutely or relatively short ramus and body of the mandible and not with an arrest of development in the incisor region." This would appear contradictory to our findings, especially the latter part, but it is to be remembered that Hellman's data in this instance had particular reference to conditions in the adult and not to infants. On the other hand, it must be noted that our near-adult (IVA) and adult (VA) groups also manifested some shortening, although not much, in the upper height of the face, as compared with cases possessing normal dental occlusion. With regard to height of ramus, according to the face profiles and judging from the position of gonion, it also appears more or less shorter in our open-bite groups.

TABLE XI

DESCRIPTIVE SUMMARY OF THE DIVERGENCE FROM NORMAL IN THE FACIAL PROFILES

| TYPE OF OCCLUSION | DENTITION STAGE | FACE LENGTHS | | |
|---|---|---|---|---|
| | | UPPER | LOWER | TOTAL |
| Poor normal | IIIA | mod. longer: prosth. lower | n. same: infrad. and menton lower | mod. longer |
| | IVA | much shorter: nasion lower, prosth. higher | sl. shorter: both infrad. and menton higher | much shorter |
| Good abnormal | IIIA | sl. longer: prosth. lower | n. same: infrad. and menton lower | mod. longer |
| | IIIC | same | sl. shorter: menton higher | sl. shorter |
| | IVA | near same | sl. shorter: menton higher | sl. shorter |
| | VA | mod. longer: prosth. lower | n. same: infrad. and menton lower | sl. longer |
| Open-bite | IIA | much shorter: prosth. higher | sl. shorter: menton higher | sl. shorter |
| | IIIA | much longer: prosth. lower | n. same: infrad. much lower, menton also | much longer |
| | IVA | mod. shorter: prosth. higher | mod. shorter: infrad. lower; menton higher | sl. shorter |
| | VA | mod. shorter: prosth. higher | n. same: infrad. sl. lower, also menton | sl. longer |
| Edge-to-edge bite | IIIA | mod. longer: nasion higher; prosth. lower | n. same: infrad. lower, also menton | mod. longer |

An attempt is made in Tables XI-XVI to summarize the preceding results. This summary in tabular form will be especially helpful, it is felt, in apprehending at a glance, in any single form of malocclusion, the type of divergence from the normal through all the dentition stages. The divergence from the normal was in every instance determined from the face profiles.

### RATE OF GROWTH

One other phase of the problem will be briefly considered. In our analysis of changes in the face of individuals with normal occlusion and abnormal occlusion, the latter regardless of type, we noted rather similar rates of growth in both. Such similarity has likewise been observed in the growth of the dental

TABLE XII

DESCRIPTIVE SUMMARY OF THE DIVERGENCE FROM NORMAL IN THE FACIAL PROFILES

| TYPE OF OCCLUSION | DENTITION STAGE | DEPTH OF FACE | | | | |
|---|---|---|---|---|---|---|
| | | AURICULO-NASION | AURICULO-PROSTHION | AURICULO-INFRAD. | AURICULO-MENTON | MENTON-GONION |
| Poor normal | IIIA | near same | near same | mod. less | near same | sl. longer; gonion position near same |
| | IVA | much greater | near same | mod. less | n. same, though menton higher due to shorter length | gonion point higher |
| Good abnormal | IIIA | mod. greater | sl. less | sl. less | sl. greater | gonion mod. above and sl. in front of normal |
| | IIIC | near same | sl. less | near same | mod. less | gonion position same |
| | IVA | same | sl. less | mod. less | mod. less | gonion sl. back due to lack of depth in front |
| | VA | same | sl. less | mod. less | mod. less | gonion sl. higher and farther back |
| Open-bite | IIA | near same | mod. greater | near same | mod. less | whole body back; gonion also higher |
| | IIIA | sl. greater | near same | near same | sl. less | gonion same; body longer |
| | IVA | near same | much less | much less | mod. less | gonion point sl. above and back |
| | VA | same | near same | near same | mod. less | gonion higher and farther back |
| Edge-to-edge bite | IIIA | sl. greater | mod. less | near same | near same | gonion point sl. higher |

TABLE XIII

DESCRIPTIVE SUMMARY OF THE DIVERGENCE FROM NORMAL IN THE FACIAL PROFILES

| TYPE OF OCCLUSION | DENTITION STAGE | FACE LENGTHS | | |
|---|---|---|---|---|
| | | UPPER | LOWER | TOTAL |
| Excessive | IIA | same | sl. shorter | sl. shorter |
| | IIIA | much longer: nasion sl. higher, prosth. much lower | n. same: infrad. mod. lower, also menton | mod. longer |
| | IIIC | sl. longer: prosth. lower | n. same: infrad. sl. lower, also menton | near same |
| | IVA | near same | mod. shorter: infrad. sl. higher; menton much higher | mod. shorter |
| | VA | mod. longer: prosth. lower | sl. shorter: infrad. lower | near same |
| Class II (Angle's) | IIA | mod. longer: nasion higher; prosth. lower | near same | near same |
| | IIIC | near same | sl. longer: menton lower | sl. longer |
| | IVA | sl. longer: nasion higher | n. same: infrad. higher, also menton | mod. shorter |
| | VA | n. same; nasion lower, prosth. lower | sl. shorter: infrad. lower | near same |
| Retrusive bite | IIIC | much longer: prosth. lower | much shorter: infrad. lower | sl. shorter |
| | IVA | sl. shorter: nasion lower | near same | sl. shorter |

TABLE XIV

DESCRIPTIVE SUMMARY OF THE DIVERGENCE FROM NORMAL IN THE FACIAL PROFILES

| TYPE OF OCCLUSION | DENTITION STAGE | DEPTH OF FACE | | | | |
|---|---|---|---|---|---|---|
| | | AURICULO-NASION | AURICULO-PROSTHION | AURICULO-INFRAD. | AURICULO-MENTON | MENTON-GONION |
| Excessive | IIA | same | same | mod. less | much less | gonion higher and back |
| | IIIA | sl. greater | near same | sl. less | near same | gonion position n. same; body longer |
| | IIIC | sl. less | sl. less | n. same | mod. less | gonion in front of norm; body shorter |
| | IVA | sl. less | sl. less | much less | sl. less | gonion point higher; body shorter |
| | VA | near same | much less | much less | mod. less | gonion point n. same; body shorter |
| Class II (Angle's) | IIA | sl. greater | sl. greater | sl. less | sl. less | gonion farther back; body longer |
| | IIIC | sl. greater | sl. greater | sl. greater | sl. greater | gonion farther back; body longer |
| | IVA | much greater | same | mod. less | mod. less | gonion point higher; body sl. shorter |
| | VA | mod. less | mod. less | mod. less | sl. less | gonion pt. in front of norm; body shorter |
| Retrusive bite | IIIC | sl. less | much less | mod. less | much less | gonion above and back; body shorter |
| | IVA | much less | much less | mod. less | much less | gonion sl. above and back; body shorter |

TABLE XV

DESCRIPTIVE SUMMARY OF THE DIVERGENCE FROM NORMAL IN THE FACIAL PROFILES

| TYPE OF OCCLUSION | DENTITION STAGE | FACE LENGTHS | | |
|---|---|---|---|---|
| | | UPPER | LOWER | TOTAL |
| Retrusive bite | VA | n. same: nasion lower, prosth. lower | sl. longer: infrad. much lower, also menton | much longer |
| Class III (Angle's) | IVA | mod. longer: prosth. lower | sl. longer: infrad. mod. lower, menton much lower | much longer |
| | VA | sl. longer: prosth. lower | n. same: infrad. much lower, also menton | much longer |

TABLE XVI

DESCRIPTIVE SUMMARY OF THE DIVERGENCE FROM NORMAL IN THE FACIAL PROFILES

| TYPE OF OCCLUSION | DENTITION STAGE | DEPTH OF FACE | | | | |
|---|---|---|---|---|---|---|
| | | AURICULO-NASION | AURICULO-PROSTHION | AURICULO-INFRAD. | AURICULO-MENTON | MENTON-GONION |
| Retrusive bite | VA | mod. less | much less | much less | much less | gonion higher and back; body shorter |
| Class III (Angle's) | IVA | sl. less | markedly less | sl. less | mod. less | gonion higher and back; body longer |
| | VA | sl. greater | sl. greater | much greater | markedly greater | gonion much in front, sl. below; body sl. longer |

arches, also when abnormal occlusion, regardless of kind, was compared with normal occlusion. We have always suspected, however, as already indicated, that such similarity was due primarily to smoothing out of differences as a result of indiscriminately combining all forms of malocclusion in one abnormal category. The following profiles (Fig. 11), comparing rate of growth in normal occlusion with the rate in each type of malocclusion, bears out our thesis.

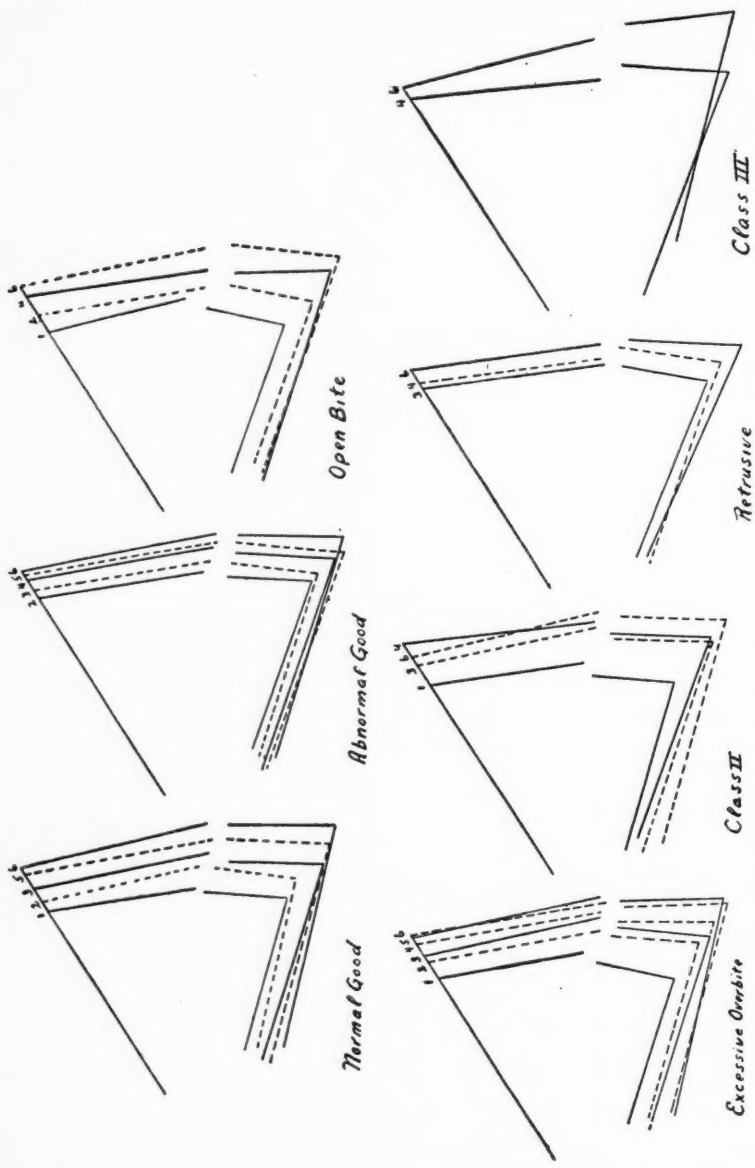


Fig. 11.—The rates of growth between two or more dentition stages as manifested in the facial profiles in normal occlusion and the various forms of malocclusion. The numbers 1, 2, 3, 4, 5, 6 refer, respectively, to dentition stages IIA, IIIA, IIIC, IVA, IVC, and VA; the alternating solid and broken lines are merely an aid in distinguishing one figure from another.

It is understood, of course, that the rate of growth is indicated in any figure by the space, or amount of increment, between two or more dentition stages.

The import of the figures is clear and need not be elaborated upon. Rate of growth is seen to differ appreciably at certain times and in the different parts of the face, in normal occlusion as compared with the various types of malocclusion, and in the several forms of malocclusion as compared with one another.

## DISCUSSION

Some further comment, it is felt, is needed regarding the use of dentition stage or period of tooth eruption as an index of physiologic age.

Aside from the question of whether the differences among the average chronologic ages within the same dentition stage were significant or not, a topic which has already received comment, we noted in the course of studying our material that individuals of 12 years, for example, would fall in the same dentition stage as subjects 18 and 21 years old. In all such cases stature was much greater in the older subjects, and facial measurements likewise were larger, although not so markedly as stature, of course. True, these instances of extreme discrepancy between chronologic and dentition ages were not many; yet, combined with cases in the same dentition stage separated by 2 or 3 years, the total number in any stage was not negligible. In other words, the data engendered a suspicion that the dentition stage includes too wide a range in time.

As a matter of fact, physiologic age may and does differ in different parts and organs of the body. The circulatory system, for example, often ages more rapidly than the nervous system; or the eye, in structure and function, is fully developed years before the sexual organs. In other words, instead of one physiologic age of the individual, there are probably several physiologic ages, all more or less correlated.

As regards stature, certainly it seems to us that the dentition stage is but a rough index of age or development, and that chronologic age, however inadequate, is a comparatively much more precise criterion. The matter is not so clear relative to head and face growth. There is no doubt that eruption of the teeth is closely correlated with growth of the dental arches. Too, there is little doubt that the eruption of teeth corresponds to or is associated more or less with growth in the face, although in this connection it may be well to recall that little or no growth occurs in the dental arches between 2 and 5 years[4] while during this same period marked growth is evident in the face. In short, we feel the stage of dentition is exceedingly useful as a criterion of age when chronologic age is not known, as when working with skeletal material or with primitive peoples. Regarding the head and face, however, and this despite the use of the dentition stages by ourselves, as well as with the greatest respect for Hellman's contributions on the subject, we are inclined to think that chronologic age, when available, and for the reasons already indicated, is a more accurate measure of time in relation to development.

Hellman devised and utilized the dentition stages primarily, it seems, because he found too little difference in the various diameters of the face from one year to the next. We may suggest that a biannual instead of annual period would probably have given significant differences. As a matter of fact, according to Hellman's own data, the dentition stages actually do closely approximate a two-year interval. But this is between one average age, representing a dentition stage, and the following average age, representing the next dentition stage. In any single stage, according to his data, the standard deviation indicates a range of three, four, and more years, precisely the situation noted in the present study.

One other matter in conclusion. The question may arise as to the efficacy of average values in a field in which the individual is of primary concern.

The average, it is to be recalled, is made up of observations on individual cases. Too, it is pertinent to remember that the average, in the usual sense of the term, and under ordinary circumstances,* is a measure of *central tendency.* In other words, in a representative sample of the population the average length of face, for example, should occur or be closely approximated in a major portion of the individuals comprising the population. Furthermore, the number of individuals with face length differing from the average will be less and less as the difference deviates in increasing measure (plus or minus) from the average.

It must be obvious from this, therefore, that the average is applicable to the individual so far as any individual under consideration is more likely than not to approximate the average dimension under consideration, and, perhaps of more import, will probably conform to the tendencies or trends ascertained by the help of averages. Knowledge that growth, for example, of the face of an individual in a given time is more likely to be in a certain direction and more or less to a certain extent, as determined by the aid of averages in a cross-sectional study, seems to us a positive contribution toward more intelligent diagnosis and treatment, whether in orthodontia, medicine, or whatnot.

This does not, of course, imply indiscriminate use of such or any kind of information in every individual case. The good judgment of the individual practitioner must be relied upon in any case of treatment, but, and this is our only point, this judgment can be more intelligently and reliably applied when based on previously and quantitatively determined information.

To be sure, as has been intimated, the average in order to be valid must be based on an adequate sample of the population. One is seldom sure of this prerequisite and for this reason, as well as to check up on errors of observation, more than a single study on the same problem is essential before conclusions can be accepted as demonstrated.

The function of the average is limited to indicate central tendency, as noted, and gives no inkling of the kind of distribution or degree of variability of the character under consideration. This latter point is manifestly important, perhaps as necessary to know as central tendency. In group studies the minima and maxima will give the range of variation, or a more precise measure is the standard deviation which gives the limits within which, in a normal distribution curve, the middle 68 cases out of 100 will most likely fall. There can be little doubt, however, that elucidation on degree of variability or extent of individual variation can be better attained by study of the same group of individuals over a period of years. This method, the so-called longitudinal, obviously requires much time before information is made available. In the meantime group or cross-sectional studies, quantitatively presented and analyzed, can yield valuable information on type and extent of trends, and in some measure also on the degree of variability that may be expected, as well as,

*The ordinary circumstances mentioned have reference to the usual type of dispersion of physical and biologic phenomena; namely, a normal distribution curve which is a figure conforming to an expansion of the mathematical binomial.

indeed, perhaps giving direction and purpose to a longitudinal study of the same problem. As was ably pointed out in a recent symposium* on this very subject, each method, cross-sectional and longitudinal, is useful and necessary in research; in fact, each is really complementary to the other.

These comments on method were indulged in because some opposition has recently been heard from orthodontists on the use of the cross-sectional method of investigation and its handmaiden, statistics. We felt that some explanation as to what we deemed were the merits and limitations of this method might expel or minimize any misunderstanding on this subject, especially with reference to our own present contribution.

SUMMARY

The problem was to investigate the relationship of growth in the face and head with the concomitant type of dental occlusion. Toward this end anthropometric measurements on face and head, and observations on dental occlusion, were made on groups of 50 native-born Jewish males at 2½ to 3½ years, and every other year thereafter until and including 20½ to 21½ years; a group of old men, averaging 74 years in age, was also examined for effects of senility. The data were organized according to stage of tooth eruption and with regard to type of dental occlusion. The results were as follows:

1. Completion of eruption of the deciduous dentition occurs somewhat earlier in abnormal than in normal occlusion; conversely, the permanent first molars seem to erupt earlier in normal than in abnormal occlusion. The permanent second molars, however, also appear earlier in abnormal occlusion. Practically no difference in time is evident in eruption of the third molars in either normal or abnormal occlusion.

2. Comparing the various dimensions of the head and face with respect to normal and abnormal occlusion, little or no difference was noted in the head except maximum width and width of the forehead (minimum frontal), each of which is appreciably narrower in abnormal occlusion cases in early childhood. In the face, the widths are also generally narrower in cases with abnormal occlusion, especially in early childhood (stage IIA); total length and upper length of face are longer in abnormal occlusion in the later dentition stages; lower face length, however, does not seem appreciably different in abnormal than in normal occlusion at any time. Depth of face at all levels is shorter, especially in the later stages, in abnormal occlusion.

3. A statistical test whether the differences between cases with normal and abnormal occlusion were significant or not revealed that the above noted tendencies were significant. In other words, during the process of growth differences in the head and widths of face, noted in early childhood between cases with normal and abnormal occlusion, became largely eliminated; whereas lengths of face and especially auricular depths to mouth became more differentiated, the face becoming significantly longer in height and shorter in depth in cases with malocclusion.

*The 1936 meeting at Washington of the Society for Research in Child Development.

4. Profiles of the face were constructed according to the mean dimensions at the various dentition stages. These demonstrated graphically the above mentioned tendencies in the face, as well as greater growth in the mandibular than maxillary region in length and depth, in both normal and abnormal occlusion; greater facial prognathism in the adult is indicated thereby. The relative position of gonion point to the ear hole is remarkably stable in the course of growth, regardless of type of occlusion.

5. The consequence of old age in the face is a shortening in length and depth, primarily due to loss of teeth and alveolar absorption. But most striking is a considerable shift forward of the whole mandible with jutting out of chin.

6. Total increment between stages IIA and VA is on the whole not much different in normal than abnormal occlusion. There is, however, much difference with respect to character considered: total relative growth is least in the head, more in width of face, and most in length and depth of face.

7. Relative variability is greater in normal than in abnormal occlusion in early childhood, except depth of face in which the converse is true; with increasing age, however, there is a definite trend toward greater variability in abnormal than in normal occlusion. In both normal and abnormal occlusion, variability absolutely tends to increase with age whereas relatively it decreases. Relative variability is generally least in the head, more in depths of face, and most in widths and lengths of face in this order.

8. To combine all forms of malocclusion into one abnormal occlusion class, it was maintained, tends to obscure important differences; hence the material was reorganized according to various types of malocclusion.

9. Dimensions of the head now indicate: (a) marked divergences in length, width, and height, in groups with various types of malocclusion; (b) a tendency for the differences among each of the various occlusion types to diminish in the later dentition stages; (c) divergence from normal tends to be in one direction, plus or minus, especially in some forms of malocclusion.

10. Face widths in the various forms of malocclusion likewise manifest substantial deviation from conditions in normal occlusion, as well as each compared with the other. Direction of divergence from normal differs in the widths taken at the various levels of the face, albeit in the same type of occlusion and at the same dentition stage.

11. The divergence in length and depth of the face in the several types of malocclusion from conditions in individuals with normal occlusion is: (a) upper face length is generally longer in all forms of malocclusion except perhaps the open-bite in which the tendency is shorter length; (b) lower face length tends to be either alike or somewhat shorter except in the late dentition stages of retrusive bite and in Angle's Class III form of malocclusion, in which it is longer than in normal occlusion; (c) auricular depth to nasion is generally the same or somewhat greater in all but the retrusive bite form of malocclusion in which this depth is definitely less; (d) auricular depth to prosthion tends to be less except in Angle's Class II type of malocclusion which mani-

fests generally greater depth; (e) auricular depth to infradentale definitely tends to be less except in Angle's Class III form of malocclusion in the adult stage, in which this depth is much greater; (f) auricular depth to chin or menton is likewise definitely less except in Angle's Class III in the adult, in which it is also markedly greater; (g) depth of mandible and position of gonion differ with dentition stage and type of occlusion. All these differences, even when in the same direction, vary more or less in extent, evidently as a result of differences in rates of growth.

12. Chronologic age, when available and its use is practicable, is deemed a better criterion than dentition stage in studies of growth of the face in general.

The task of accumulating and preparing the data comprising this study was a heavy one, and one in which indispensable aid was received from many sources. We are indebted to the Board of Education of the City of New York, and especially Dr. Eugene A. Nifenecker, Director of Reference, Research and Statistics, for generous permission to work with the children in the public schools. For initial and safe guidance through the intricacies of obtaining this permission, and indeed, for generously placing the facilities of school and boys at our service, we are much obliged to Dr. Alfred A. Tausk, Principal of the Boys' High School of Brooklyn. Mr. J. B. Fish, head of the Biology Department in this great institution, was of inestimable aid in rounding up the boys. Sincere thanks are due Miss Matilda Srager, Principal of Public School 160 of Manhattan, for kind permission to examine the smaller children in her school. To Miss Mary Creed, Superintendent, and the Medical Board of the Home for Hebrew Infants in New York City, we are indebted for permitting us to measure the infants taken care of in this remarkable institution. We would like also to thank the attendant nurse, Miss Mary O'Neil, whose masterful handling of the infants contributed no little in making the observations feasible. Our thanks are due Mr. L. J. Simmonds, Executive Director of the Hebrew Orphan Asylum of the City of New York, and Dr. J. Novikoff, head of its dental department, as well as Mr. A. L. Jacoby, Executive Director of the Hebrew Orphan Asylum of Brooklyn, for their generous cooperation in obtaining the 4½ to 5½ year age group. The young men of 18½ to 21½ years were for the most part obtained at New York University primarily through the kind assistance of Professor William McTavish and his staff. To the Medical Board of the Home of the Daughters of Jacob in the City of New York, and its capable staff of nurses, we are much indebted for their kind permission and help in obtaining observations on an old-age group.

Our acknowledgments are also due Mr. A. Magilef, an aid in the Division of Child Research, who had a large share, under our supervision, in the laborious task of tabulating the great mass of data involved in the present study.

Finally, we wish to express our appreciation to all the infants and children and men who more or less willingly suffered themselves to be examined.

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# ORTHODONTIC EDUCATION FOR THE UNDERGRADUATE

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THERE is much to learn in a study of the history of orthodontia with its slow development as an adjunct of general practice, and its later, more rapid progress. Modern orthodontia, dating from about nineteen hundred, has become more and more complex, including a greater concept of biology, and emphasizing the study of growth and development, in addition to the old purely mechanical problems. This period has seen the rise of the specialist, who made orthodontia his life work. The greatest progress has been made in this way, and one feels that further progress will be achieved mostly by the continued concentration of energies in specialization. Orthodontic teaching originally delegated to the professor of prosthetics, and developing at a slower rate than its practice, has now reached the point where it is recognized that a man hoping to practice orthodontia exclusively should have a full year of instruction in the graduate department of a university.* In addition to this, some time spent in association with an orthodontist would help considerably, especially in everyday, practical aspects of practice. This does not mean at all that the older men now in orthodontic practice must be considered as unprepared. They grew as the knowledge of orthodontia grew, and their preparation, mostly by a system of trial and error as they went along, was long and arduous. Today, with a larger body of orthodontic knowledge, increasing year by year, teachers feel that those undertaking practice without thorough preparation are deliberately handicapping themselves. In the days when orthodontia was known only as a comparatively simple problem—short courses and preceptor work appeared sufficient. Today the requirements of practice are greater than is apparent to the layman or the dentist or even to the average dental educator.

Present-day undergraduate courses in orthodontia have not been standardized nearly to the same extent as other subjects in the dental curriculum. We have everything advocated—from the teaching of the barest essentials to the radical extreme of starting the specialized training in the freshman year, and finally qualifying the graduate only in the practice of orthodontia. I do not believe that most dental educators would want to relegate orthodontic teaching to a minimum. It is rapidly becoming recognized that the course in orthodontia rounds out a general dental education. It gives an emphasis to the real underlying purpose of dentistry, namely, the establishment and maintenance of the health, usefulness, comfort and beauty, of the masticatory apparatus. It stresses the modern point of view of prevention. It provides a new angle of thought to—and ties up closely with—prosthodontia, exodontia, operative dentistry (especially for children) and periodontia. Neither do I believe dental

Presented to the American Board of Orthodontia.

*Resolution passed by the New York Society of Orthodontists, November 21, 1933. *Dental Items of Interest*, page 237, March. 1934.

educators to be in accord with a plan that would make a man a specialist in any branch of dentistry without becoming first a dentist. There does not seem to be any valid reason why the requirements of specialization in orthodontia should be different from those of any other specialist in medicine or in dentistry. To be most useful, he should be able to visualize his problems first in the general field of dentistry and then according to his specialized training. There is a tendency for too much specialization to result in a narrow-minded perspective. An orthodontist should be a good dentist first and a specialist afterward. Many of our best orthodontists practiced general dentistry for some years before limiting their practice. This has often been suggested as an ideal way to prepare for specialization. However, with the better teaching of general dentistry in the schools, I do not agree that this is necessary. The specialist can keep up on general dentistry by attending dental society meetings. Between the extreme minimum and the extreme maximum amount of orthodontia to be included in the curriculum, there is surely some happy middle ground. Considered from all angles, our problem consists essentially of just two points—the determination of how much orthodontia the undergraduate should have and how it should be given; in other words, the amount and the character of instruction. It seems to me that these points may be determined by discussion of the following: (1) the need for undergraduate instruction, (2) the ability of our schools and their faculties to give instruction, (3) the ability of our undergraduate students to absorb such instruction, (4) the time that may be allowed for such instruction in the curriculum.

The need for undergraduate orthodontic instruction is obviously determined by the necessity for orthodontic services. Although accurate statistics are not yet available, school surveys indicate that from 50 to 80 per cent of our children are in need of orthodontia, with a very small percentage under treatment. There are only some six hundred specialists in orthodontia practicing in this country. Probably more cases are being treated by general practitioners than by orthodontists. Even so, there remains a situation wherein the overwhelming majority of our children who need orthodontic service are not getting it. Some 90 per cent of these children are in an economic stratum which would prevent them from paying for such services if these services were available. Orthodontic service cannot be performed very cheaply in either general or limited practice. Mass production ideas have even less bearing upon orthodontic treatment than they do upon treatment by the physician or the general dentist. Each case is individual, requiring its own painstaking effort. Directing this effort, must be an operator trained not only in the special mechanical technic required, but with a mind well grounded in the biological aspects, and with the seasoned judgment of experience. The general dentist cannot treat these cases cheaply; in fact, it is doubtful that he can treat them as cheaply as the orthodontist. He is usually without the specialized office organization for highest efficiency in this work. He is comparatively less skilled in the technical procedures, and would therefore take a longer time in appliance construction and adjustment. With comparatively less experience, his judgment is not likely to be so good and his treatment time would be lengthier. The volume of cases

would be lower, suggesting a higher fee per case. In fact, it has been my observation that most cases in larger cities undertaken by the general dentist are selected only when the patient can pay as high or even a higher fee than the orthodontist would charge. This is only natural because of the reasons stated above. Of course, this would not apply in communities in which the orthodontist is not immediately available. Here, orthodontic treatment is more or less forced upon the conscientious dentist, and he must do something whether the fee be high or low. The problem of corrective orthodontic treatment for the millions of children in the lower economic strata is only one of the many unsolved social questions of the day. We have not been able to solve even the question of general dental treatment for these children. While the number of children in need of general dentistry is slightly larger than those needing orthodontia (around 90 per cent in all), the problem is simpler due to the difference in time element and the vastly greater number of trained operators. However, if we believe in orthodontia as a health service, we must look forward to solving this problem.

So much for the need for orthodontia. How about the demand? Evidently the demand is greater now than the orthodontists can meet; but with increasing laity education the demand will grow—in fact, is growing—year by year. There will be more and more people realizing the importance of treatment and willing to pay for it. As economic conditions become stabilized, this demand will assume hitherto unthought of proportions. Any one who has been connected with an orthodontic clinic feels the advancing interest in orthodontia. How are we going to satisfy this demand? Increasing numbers of orthodontists may take care of it to some degree. Does the answer lie in the practice of orthodontia as an adjunct of general dentistry? I think not. There are certainly some men in general practice who successfully handle at least the simpler cases of malocclusion. These men have invariably put in a great deal of time in postgraduate study and in learning technic. The difficulty is that the number of men willing to do this is limited, and the "simple" cases are difficult to define. I cannot believe that in most cases orthodontic treatment can be successfully handled along with general practice. Orthodontia is a stern taskmistress. She will brook no rivals. Full time is too little time to devote to her needs. There is far too much poor orthodontia done now. We need better orthodontia rather than poorer. How much harm is being done in the name of orthodontia today is not known, but there is a reasonable suspicion that there is a great deal. Most orthodontists hesitate to look back critically over some of their first fifty or one hundred cases. The problems are so diversified and often obscure that it seems that only long experience can teach the pathway to first-class treatment. It is my belief that the average dentist, without a great deal of special work and study (and without the experience of many cases), does himself more harm than good by "going into" sporadic orthodontic treatment. If the orthodontist, specializing in his own field, finds pitfalls and failures—even dangers—confronting him, how much more so the dentist in general practice? Nefarious laboratory advertising has done much to lure general dentists into the field of orthodontia. They promise to do what no highly qualified orthodontist in the wildest flight of

his imagination would ever think of doing. Without x-ray pictures, without history, without seeing the patient—with only a set of casts—they would diagnose, design and make appliances and even outline treatment for any case of malocclusion. This is, no more or less, a plain unvarnished insult to the intelligence. Orthodontic cases are not diagnosed that easily. Appliance design is not according to a routine pattern. Treatment is not standardized, nor do cases respond with standard results. Most important of all, treatment is not a purely mechanical problem. It is primarily a matter of directing growth and development into normal paths. Does any one think these laboratory technicians understand this important point? If they do, can they direct this growth by mail? I feel that those who state that the increasing demand for orthodontic services should be met by increasing this service as an adjunct of general practice are not fully aware of the scope of orthodontic practice. I say this with all humility, realizing my own limitations. Increasing numbers of orthodontists cannot hope to meet it for many years. I believe the answer, not only to this question of meeting the demand but to that of meeting the need for orthodontic services, can be at least partially met by the practice of prevention.

Preventive orthodontia must be sharply distinguished from corrective orthodontia. It may be quickly, easily and efficiently practiced by the properly educated (and oriented) dentist. How different from corrective orthodontia! It is the only possible hope for the children of our lowest financial stratum. It would be immediately available and an inestimable boon to those who could pay for simple dental operations, but not for long drawn-out treatment. It would even be a blessing to the others who could pay, but to whom it might save long, tedious months and years of wearing appliances. What are the facts? How much malocclusion is preventable? Again statistics are unreliable, but 50 per cent would certainly not be too high an estimate. Frederick B. Noyes* says, "I am sure that three-fourths of the cases which I have had under treatment could have been prevented by the general practitioner." Walter T. McFall† states as a result of his investigations that only 17 per cent of children under proper dental care require orthodontic treatment (because of other etiologic factors). Arthur D. Black‡ writes, "How much would all other dental services be simplified if every dentist would consider it a part of his duty to educate mothers in regard to the many things which tend to prevent irregularities of the teeth!" Also,§ "An extended experience with carefully kept records shows the number of cases of serious malocclusion to be comparatively small if there is good care of the mouth during early childhood." L. M. Waugh¶ writes, "It is strongly emphasized and frequently reiterated that the dentist can prevent the need for at least 33 per cent of corrective treatment." Other authorities could be quoted ad infinitum. It seems to be one point upon which all orthodontists and many interested dentists agree. With estimates varying from 33 per cent to 83 per cent, we seem safe in our statement that around 50 per cent of malocclusions could be prevented by early and efficient general dentistry.

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§Transactions Seventh International Dental Congress 2: 1339, 1926.
¶Waugh, L. M.: Dental Cosmos, page 509, May, 1930.

There seems little doubt that this percentage could be materially increased by properly educating our undergraduates in preventive orthodontia. The general dentist is getting to see his child patients at a very early age these days, certainly before the orthodontist usually sees him. The success of this program depends not only upon better dental education, but upon more and better laity education along these lines. However, these things are feasible and by far the most reasonable solution of our problems. What are some of the steps to be taken by the general practitioner? Herbert Pullen* lists them as follows: (1) retention of space for prematurely lost deciduous teeth, (2) extraction of too long retained deciduous teeth, (3) restoration of full contour and contact of permanent (I would add, deciduous) teeth, (4) removal of local interference in the attainment of occlusion of the teeth and in the growth of the jaws, (5) early care of the first permanent molars, sulci and mesial, (6) detection of deleterious habits, (7) early reference to rhinologist or to the orthodontist, (8) frequent prophylactic treatment of children's teeth once a month. With these steps and some additional ones in mind, it is clear that preventive orthodontia can be included in the routine of general practice. It can be made a part of regular periodic examination and prophylaxis. It would not require either a great amount of extra time, costly materials or difficult technic. If properly handled and understood by parents, it would be a source of added income and a wonderful practice-building idea for the average dentist. It would result in that feeling of satisfaction in work well done which more than compensates the professional man for the trials and tribulations of practice. He would come to realize that in applying preventive orthodontia he would be accomplishing more for the future well-being of the child's mouth than an orthodontist could do later in months of corrective treatment. It may well be made an important part of the newer, enlightened practice of dentistry which is a far cry from the old mechanical ideas. It would do much to elevate the dentist as a professional man rather than a glorified artisan.

A man with a good theoretical background, with some clinical observation and some laboratory work designed to show methods of prevention, should be qualified to practice preventive orthodontia. But the course should be given with prevention in mind. It should stress normal development, classification of malocclusion, etiology, and direct prevention. It would still serve as a broad enough outline for the comparatively few who would want to take a graduate course in orthodontia. We seem justified in stating that the need for controlling the malocclusion in our children is very great and that the demand for services is great and growing greater—limited only by the pocketbook and to a lesser extent by the lack of public education. It is most certain that the need cannot be met by corrective orthodontia for years to come—if ever. The only reasonable method of control lies in prevention. Therefore, our undergraduate education should be shaped in amount and character to result in turning out men qualified to apply preventive orthodontia.

There are, however, other considerations which have a bearing on and will continue to influence the amount and the character of such instruction. Many

*Pullen, Herbert: Dental Cosmos, page 475, May, 1933, discussion of paper by Frank Casto.

schools have not the facilities nor the money to devote to ambitious orthodontic programs. If the undergraduate is to do clinical work, he requires much expert supervision, and many more instructors would be required. In smaller university towns, these men would not only have to be orthodontists, but full-time men as well. There would not be an opportunity for exclusive practice in such towns for all of them, as there might be in large cities. The running of the orthodontic clinic requires special clerical work and special facilities which would increase the expense. Many schools have tried to run an orthodontic clinic without special teachers and equipment, and have given it up as a precarious undertaking—both from the school's standpoint and from that of the welfare of the public. In large, wealthy institutions in which the conditions are just right, it may be successful. However, even in these institutions, may not the superficial information imbibed suggest to the students that they are now ready and well qualified to practice any and all forms of corrective orthodontia? This type of course would be splendid for the few who eventually take a graduate course in orthodontia. It would be fine if these men knew what they wanted and would all go to such institutions. We are considering, however, the average student in the average dental school.

As to the ability of undergraduate students to absorb a great amount of orthodontic instruction, I am doubtful. Not that they are not old enough or have not had enough basic courses (for they now have these things in the various five- and six-year courses), but they have too much general dentistry on their minds and are not enough interested. Any one who has had experience in trying to teach even the fundamentals of orthodontia to junior or senior dental students will realize that going much farther than that would be difficult. Better to attempt the probable and perfectly possible task of limiting the course to essentials. It is not alone the apathy of the students toward orthodontia. That may be overcome by an interesting presentation and by placing the course in a position of importance in the curriculum. It is the different approach, the new angles and terminology, and a different line of thought that seems to upset the students. Due to the comparatively recent development of modern orthodontia as a science and art there are other teaching difficulties. In the fields of etiology, diagnosis, classification and treatment there are various and somewhat divergent schools of thought. These controversial ideas must all be presented in a broad way, and it is difficult for the student to arrive at definite conclusions. Frank B. Casto* says, "It is becoming increasingly more difficult to present orthodontia to general practitioners in a comprehensive manner." So, I would say emphatically that advanced teaching is too complex for them. Every year, for the past five years, I have been simplifying my theory course given to juniors, leaving out by now, nearly all of the treatment, mechanics of appliances, and postoperative problems, and stressing normal growth, etiology, classification and tying them all up with prevention. The classes are doing better and still can pass at most any state board examination in orthodontia. I trust that the men are also more awake to the possibilities of prevention.

*Casto, Frank B.: Dental Cosmos, page 466, May, 1933.

Any one who has tried to squeeze more time for orthodontia out of a dean of dentistry knows how full the curriculum has become. The students these days are supposed to engulf a vast deal more than formerly, and they are adding more. There seems to be only one way to gain more time for orthodontia, and that would be to add a year to the course and make it seven—which heaven forbid. The average dentist has a hard enough time now paying for his education. If the colleges were prepared to give the instruction, if they had the men to do it, if the students could absorb it, I still do not see how they could fit it in. The only possible solution would seem to be to provide electives in orthodontia. These would mostly be taken by those who aimed at specializing. For them it would be fine. Those who took the electives would have to eliminate some of their general dentistry. Secretly, I have an idea that would be fine too, but probably few members of the dental faculty would share my view. Therefore, I would say that the time allowed for undergraduate orthodontic instruction is now, and will be, somewhat limited by the increasing number and complexity of the courses in general dentistry.

It must be obvious from what has been said that the greatest need is for general practitioners who can apply preventive orthodontia. These men can do the greatest good for the greatest number. They could render the problem of caring for the vast number of children needing orthodontia, but economically unable to get it, within hope of solution. To summarize—the facilities and the abilities of the ordinary dental school to offer extensive orthodontic undergraduate teaching are limited. The ability of the student to grasp and absorb elaborate teaching is limited, especially as his ability is again limited by his apathy toward the subject. The time that could be allowed for orthodontia in a crowded curriculum is also limited. What then should be our plan for orthodontic undergraduate instruction?

The main problem in teaching preventive orthodontia lies in orienting the student's mind so that he can grasp conditions as they are: i.e., that we cannot teach him to be an orthodontist, that as a general dentist he cannot ordinarily be expected to practice orthodontia, that there is a vast field of preventive orthodontia opened up to him in which he can play an important rôle without further graduate instruction. We must sell him the idea of general preventive dentistry and then direct his attention to those things in orthodontia which he can make his own and put into practical use every day in his office. I always stress in this connection the objects of the course: (1) the rounding out of his general dental education, (2) the ability to advise the parents of his child patients on orthodontic problems, (3) the ability to practice preventive orthodontia, (4) the ability to talk intelligently to physicians, nurses, other dentists and laymen on the subject, (5) the necessity for knowing the essentials to obtain his license to practice dentistry. Other points mentioned are the practice-building possibilities for the young man who can handle children intelligently (including the growth and development of a good occlusion), the fact that parents are becoming orthodontia minded and can sense a lack of knowledge in that subject, that the dentist must know the essentials to avoid making embarrassing mistakes, that he must be shown that he will attain to a lasting satisfaction in his work

in the realization that he is doing more for the child in practicing preventive orthodontia than a whole battery of orthodontists can do after the condition has become complex, and that he is practicing a modern profession rather than that of the artisan or mechanic. With these points firmly fixed in mind, we are in a position to obtain more understanding and whole-hearted cooperation from our students. They realize that they are not going to be led through tangled mazes and theoretical bypaths, but are going to be given a condensed practical course which they can use.

How can we best get our essentials across to them? How much time, and what kind of teaching? I believe that we can cover the subject in one year, the junior year—with some practical application of our principles in the senior year. The junior year is selected for many reasons, the most cogent being: the student is mature enough to grasp the new viewpoint that will be presented to him and he has had a grounding in general dentistry. After a year of discussion he still has time to try out preventive orthodontia in his senior clinical dentistry. The junior year seems to tie into the rest of the curriculum with the least difficulty (at our school certainly, and from observation, probably at most schools). The course in the junior year should include a full year of lectures, one hour a week. The subject matter should include: (1) a historical background of orthodontia—brief and to the point, (2) a study of the aims and benefits of orthodontia, (3) a study of the growth and development of the normal, including the "forces of occlusion," (4) classification and diagnosis, to impress upon the student the diversification of cases, and to help later in pointing out what types of cases must be treated *early* in life, (5) etiology, which should be emphasized and made practical, and, finally, (6) prevention—which should tie up not only with all that has gone before but with all of the other general dental courses that touch orthodontia so closely.

The lectures are elucidated by clinical demonstrations. It should not be too difficult for any dental school worthy of the name to employ enough orthodontists who can devote a half day a week to the college orthodontia clinic. If only a dozen cases are carried on at a time, the students can gain the practical viewpoint that is so necessary to their proper grasp of our problems. The clinic may properly include much preventive work among its cases, stressing the differences in technic and time and quick results from those of corrective treatment. A group of not more than a dozen students should be allowed in the clinic at one time, and these should be made to comprehend every step of the procedure. All treatment is carried on by qualified orthodontists who may be assisted by graduate students (if there are any), but always under supervision. The clinical observation does not include anything but *observation* for the undergraduate.

At the same time the juniors should have a laboratory course which should include the essential technic for practicing preventive orthodontia. Impressions and casts may be included; then band making, and the making of space maintainers. Anti-thumb sucking devices may be included. Muscle exercises may be taught either in this division or in the clinic, or both. Devices to prevent mouth-breathing, to develop the arch by exercise, and other preventive devices

that may be properly included in a laboratory course can be offered by those competent to teach them. We are learning more and more about preventive orthodontia, and this part of the course should grow in interest and efficiency.

In the senior year, the department of orthodontia should act in the rôle of advisor to the student in the orthodontic aspects of his clinical dentistry. Much can be done in teaching the student to recognize both incipient and well-defined types of malocclusion. Also here he can start in the actual practice of preventive orthodontia. In large schools, with sufficient endowment and available faculty, electives may well be given in orthodontia for those students evincing a special interest or ability. It is to be hoped that *all* schools in the future may be able to do this. Also, in the larger schools, the graduate orthodontia department can serve the undergraduate student by furnishing means for larger clinical observation and possibly in other ways. I believe that the above outline, if followed reasonably well, would place the student in as good a position to do preventive orthodontia intelligently, as he would be to do any other form of dentistry. Further development into proficiency can come only with practical experience. However, the graduates would be in a position to go forward and grow and develop into important factors in the field of orthodontic service to our children.

The problem of controlling the malocclusions of some ten to eighteen million children is stupendous. It seems beyond the possibility of solving; yet, if we believe in the right of every child to a healthy body, if we believe in orthodontia, we must consider this problem. The element of time, the materials, the lack of experienced operators are insurmountable barriers to wholesale corrective treatment. The first step in solving the problem and the only one that seems in any way practical lies in prevention. If we can eliminate 50 per cent of these malocclusions by preventive measures, we have cut the problem in half. Preventive measures require little time and material, and every dentist could be a trained operator. Outside of a few exceptionally interested dentists and those whose rural location forces them to become somewhat qualified in orthodontia, the average dentist can do more good both for himself and for his patients in practicing preventive orthodontia than by unqualified attempts at corrective measures. Let us plan to meet at least partially the present and future problem of malocclusion in our children by strengthening undergraduate orthodontic education along preventive lines.

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# USE OF ARTIFICIAL CINGULA WITH OTHER APPLIANCES

CLIFFORD G. GLASER, D.D.S., BUFFALO, N. Y.

THE artificial cingula are small attachments soldered to the lingual surfaces of incisor bands to represent an extended development of the natural cingula of the tooth. They are so adapted as to act as guides of the incisal bite.

In deep bite cases there must be an extension of the attachments from the incisor bands even beyond the gingival line and upon the gum tissue. Then as the case progresses they are replaced with shorter extensions until the incisal bite has been changed to a point where direct attachments can be

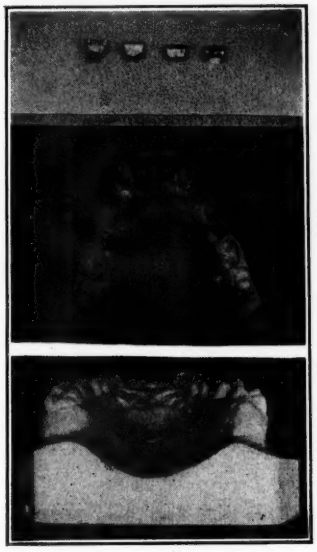

Fig. 1.

Fig. 2.

Fig. 3.

Fig. 1.—Incisor bands with artificial cingula attached showing four stages of attachment.

Fig. 2.—Cast showing two stages of attachment and positions on teeth. At each stage both incisor bands have the same height of attachment.

Fig. 3.—Cast showing two advanced stages of attachment in which the cingula are attached directly to the band instead of having any extension.

made without extensions. These also are replaced with others nearer the incisal edge until the desired depth of the incisal bite is established.

It is my observation that no appliances should be fixed by attachment to these teeth except the ordinary contact of a labial arch wire. The mandibular teeth are supported by a lingual arch, and intermaxillary elastics are used.

It is imperative that we do not allow the patient to use the tips of these artificial cingula for incisal biting, lest we be defeated in our intended purpose to use them as guides.

Presented before the New York Society of Orthodontists, New York, N. Y., Nov. 23, 1936; and at the Thirty-Fifth Annual Meeting of the American Society of Orthodontists, Chicago, April, 1937.

The artificial cingula are constructed of wire rather than flat metal, for cleanliness, and to allow the most natural functional stimulation of the gum tissue.

Although Fig. 1 shows four steps, there may be more or less. The bite should never be opened so far that all posterior occlusion is lost. The

Fig. 4.

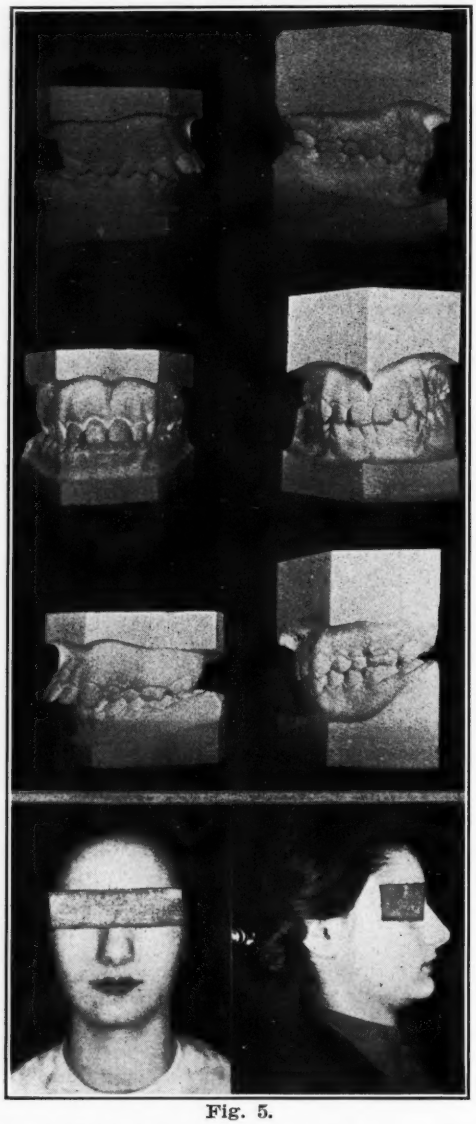

Fig. 5.

Fig. 4.—Casts of case showing change effected by use of artificial cingula.

Fig. 5.—Present appearance of face of same patient (front view). The right side of the face is longer than the left side. This is also shown in the gnathostatic cast of Fig. 4. A study of the history of this case reveals that this is the patient's natural inheritance.

patient must be allowed a fair contact of the posterior teeth in the lateral occlusions.

The lingual appliance used is one which allows a good response of the teeth and supporting tissues to functional stresses by allowing freedom of motion rather than determined fixation.

The appliance described herein is one in which there has been a consolidation of base wire and auxiliary springs. The arch wire is constructed of a small gauge wire (0.025 inch) and is adapted so that it follows the gum line from molar to molar on the lingual side of the teeth. There are four anchorage points, namely, the two first molars and the two lateral incisors or perhaps the two cuspids instead of the lateral incisors.

In the construction there are attached to the molar bands, close to the gingiva, small simplified locks which reduce the usual bulk of material necessary in locking devices. The incisor bands have small hooks attached near the gingiva, and the arch wire has a loop placed between the bicuspids on each side for the purpose of spring and adjustment.

Fig. 6. Fig. 7.

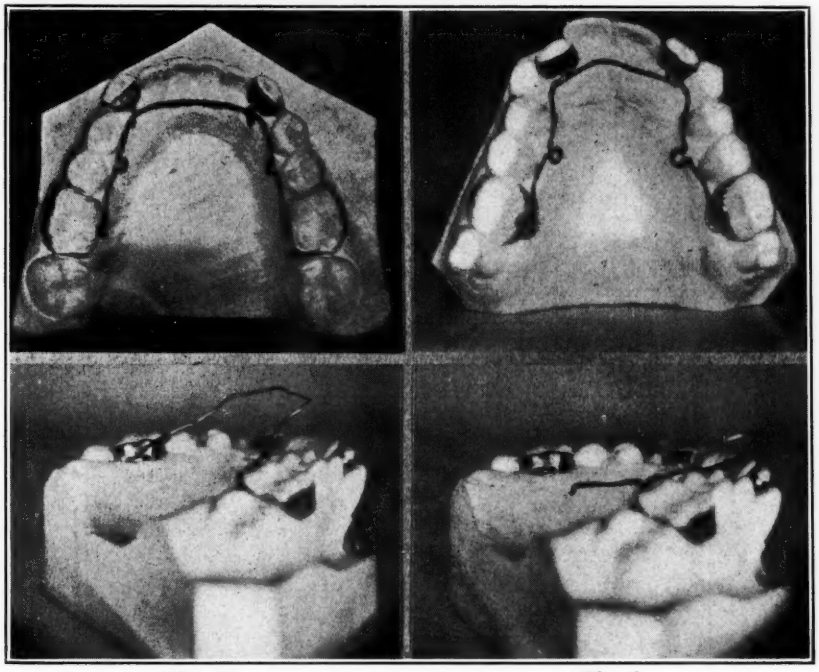

Fig. 8. Fig. 9.

Fig. 6.—Appliance used on mandibular arch, showing closeness of adaptation at the gingival line.

Fig. 7.—Same type of appliance as shown in Fig. 6 but for a maxilla. This is not an appliance used on the case just shown but is exhibited here to show the construction and operation of appliance shown in Fig. 6.

Fig. 8.—Appliance halfway in position. To show method of application or removal.

Fig. 9.—Construction of end of arch with its reverse bends to form male part of attachment. Also counterpart of lock attached to band.

The end of the arch has a sharp reverse bend (Fig. 9) which, when inserted into the lock as shown in Fig. 8 and then the anterior part of the arch brought to the incisors, locks it in place. Then the arch is sprung under the hooks on the incisor bands. The arch in place is shown in Fig. 7.

The closeness of adaptation is very necessary for its stability, cleanliness, comfort, health of the tissues, and use of a small gauge wire.

To prevent rotation of the molars a contact of the arch wire and the molar band just anterior to the molar lock must be maintained, unless such rotation is desired.

Adjustment is made by changing the form of the arch, which is sometimes quite irregular at the beginning, to a more symmetrical form. The size of the arch is increased by opening the loops.

Fig. 10.—Views of a case showing effect of use of artificial cingula with other appliances.

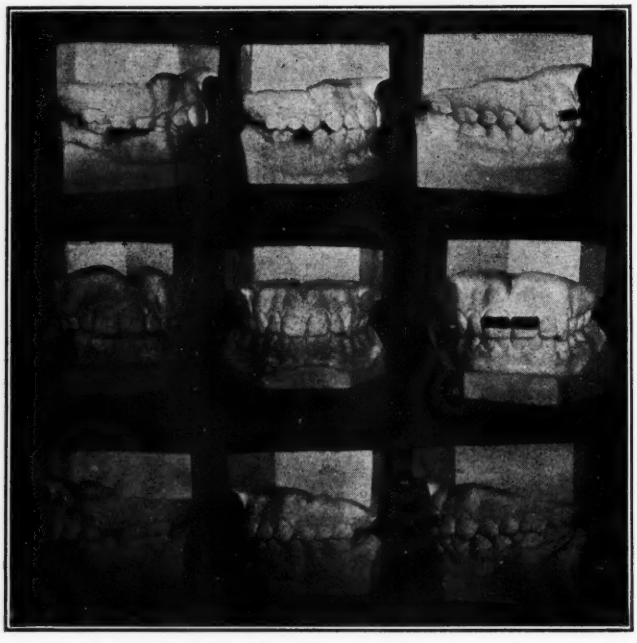

Fig. 11.—Views of a case showing three stages of change. From the second stage to the third stage no other appliances except the artificial cingula were used.

There is no positive fixation of the molars to interfere with their functional stimulus. Any desired lateral movement of the molars is accomplished by very little lateral spring in the appliance, but mostly by the end thrust of the arch wire.

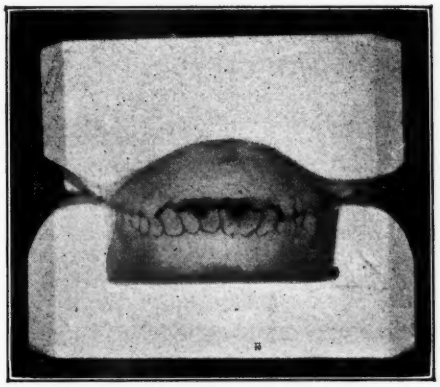

Fig. 12.—Lingual view of artificial cingula in place on cast to show how they fit parallel to surfaces of mandibular teeth when teeth are in occlusion. When teeth are set, there are no sharp projections in the mouth to interfere with proper tongue support of the anterior teeth.

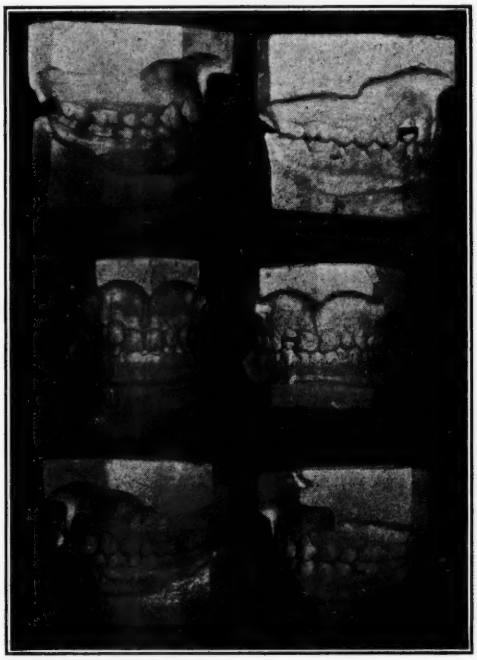

Fig. 13.—Right side of a case showing the use of the artificial cingulum on a lateral incisor as a retentive appliance. It will be noticed that this tooth was quite prominent in the early stage of development.

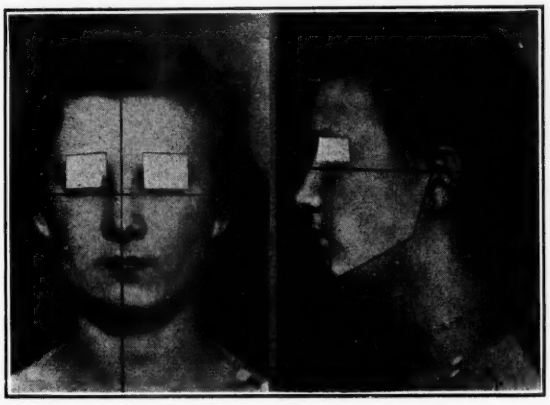

Fig. 14.—Face at present, showing a slight unilateral distal relationship of the mandible.

The wire used should not be too stiff so that it may be handled easily in adjustment. It must also not be too weak so that the arch is somewhat floppy in the loop area.

To me it seems that this appliance allows more natural function of the teeth, and therefore is most efficient when myofunctional therapy is practiced with its use.

Although Figs. 7, 8, and 9 show a maxillary arch, it is more indicated for use on the mandible.

Except in rare instances, no spring wires are attached to this appliance. After more practice with it, the spring wires will become less and less necessary.

DISCUSSION AT MEETING OF NEW YORK SOCIETY

*Dr. Glaser.*—I neglected to say that there is a labial arch used on the upper there [indicating picture on the screen] during that same time, and it is important to have that insertion so that you can connect up the different points of treatment.

*Dr. Harry E. Kelsey.*—I think it is a very fine adaptation of the idea which has been presented so often in very many different forms, and I am sure that it would prove very useful in anybody's hands who would carry it along the lines that Dr. Glaser has so well illustrated. It especially appealed to me as a control for tongue habits at the same time, and might be extended for that purpose alone in other pernicious forms of malocclusion. I thought it was a very excellent presentation.

*Dr. Glaser.*—There are one or two little points that I left out. The anterior bite needs always to be watched to see that we have stresses of function brought to bear against the lingual surfaces of the maxillary incisors more than against the bite guides; that if we allow the stresses of function to come directly in line against the mandibular incisors we defeat the purpose that we want to be effected; that we need that stimulus to come from the direction that is normal or natural that an incisal bite should bring to bear, i.e., against the lingual surfaces of the maxillary incisors and from the labial surfaces, to some extent, from the mandibular incisors. I think that is important.

# THE GRIP TUBE

ASHLEY E. HOWES, D.D.S., NEW ROCHELLE, N. Y.

I HAVE been allotted ten minutes to describe a mechanical innovation which if regarded merely as another gadget could be described in one minute. On the other hand, its application could lead to quite a lengthy discussion. I have no intention of going into any dissertation on mechanics in orthodontia, but one controversial subject must be mentioned because this new tube has to do with it, and that is the moot subject of stainless steel for orthodontic appliances.

Since the introduction of this material to our specialty I have been greatly impressed by many of its properties, but even more impressed by many of its disadvantages. It seemed to me that its great tensile strength is a most decided advantage, inasmuch as much smaller gauge wires can be employed. Its chief disadvantage is the fact that heat greatly destroys this prime superiority. I therefore felt that if there was a way of using the material without applying heat in constructing the appliance, in some parts of some appliances it would be better than precious metal. The comparative costs of the two materials I believe can be disregarded as unimportant. Stainless steel seems to me to be particularly applicable for the following uses: auxiliary springs, coil springs, and buccal arches. Its use for auxiliary springs combined with precious metal arches will not be discussed at this time except to say that I have used Dr. Varney Barnes' method of attachment for several years.

In order to use the material as a buccal arch it was necessary to devise a means of doing so without the use of any heat in its construction. This, of course, was simple enough if no attachments were necessary, but the attachments were the problem. For this purpose the grip tube seems to me to fill a great need.

It is a tube about a millimeter or a millimeter and a half long, with an open seam in it, made of hard drawn stainless steel and made in various gauges depending on the size of wire to be used (Fig. 1). For instance, if we want to make a buccal arch of stainless steel, 0.032 or 20 gauge seems to be heavy enough. A grip tube with an inside diameter to fit 0.028 or 21 gauge wire is used. One end of the piece of wire to be used as an arch is tapered very slightly and tapped into the tube. The seam in the tube allows the tube to expand to accommodate the large gauge wire but the tube grips the wire very tightly. However, after the wire is once started into the tube, by means of a modified pair of pliers, which can be made from a discarded pair of Howe pliers, the tube can be slipped along the arch to any desired position. Its grip upon the wire is such that it cannot be displaced except by means of

Read before the New York Society of Orthodontists, New York, N. Y., Nov. 23, 1936.

pliers; therefore it will withstand any orthodontic force applied. After one tube has been placed on one end of the wire, the arch is partially shaped and cut at the approximate length needed. The other end is tapered and another tube is slipped on that end. The principal point to be borne in mind is that no bends be made in the wire in the region in which the tube is to be before the tube has been put on. The arch is then shaped as desired, and the grip tubes are slid along to touch the buccal tubes if the grip tubes are to be used merely as stops.

However, if we desire to lengthen an arch by means of ligatures to the anterior teeth and coil springs on the arch, the tubes can be slipped along until there is room enough for coil springs between the grip tubes and the ends of the buccal arch (Fig. 2). Tension can be increased even without the removal of

Fig. 1. Fig. 2.

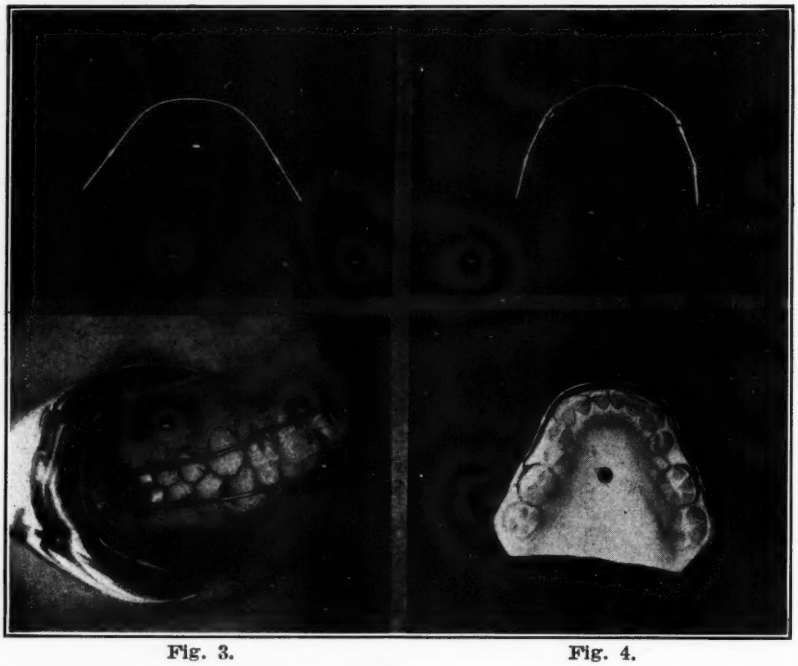

Fig. 3. Fig. 4.

the ligatures, merely by forcing the grip tube along the arch the required amount. Incidentally in changing the position of this stop, we can change it the exact amount desired without any trouble or guesswork. Fig. 3 shows such an arch in position in the mouth.

I have also had the tube made up with a little hook on one end so that a traction force can also be employed (Fig. 4). Intermaxillary hooks can be added to a buccal arch merely by soldering a hook to one of these tubes or to a seamless precious metal tube, sliding this over the arch and then placing a grip tube behind it to hold it in position. However, where intermaxillary hooks are to be used, in most cases I still prefer the precious metal arch because I generally have to add some form of attachment to such an arch. The tube is equally valuable in its application to precious metal arches when used as a stop on such an arch. The chief advantage is that it actually strengthens the arch at its

most vulnerable point, that is, where the arch leaves the buccal tube, rather than weakening it as a soldered stop does. Its position can be changed at the chair without any soldering operation, which incidentally results in saving some time. In my practice I am finding more and more use for these simple little tubes. They require no particular change in technique for the men who are using plain buccal arches for some of their tooth movements. In my hands they have proved so valuable that I felt justified in presenting such a simple idea to the Society with hopes that the tubes may prove to be an aid to someone else.

# Department of Oral Surgery

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## FIBROUS OSTEOMA OF THE JAWS

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TUMORS of the jaws have to be considered apart from tumors of bone in general. In the first place, many of them are of dental origin which precludes their occurrence in other bones. In the second place, the jaws belong to the group of bones preformed in membrane which differ considerably in growth, repair, and tumor formation from the bones preformed in cartilage.

*Embryologic Considerations.*—The maxilla and the mandible as well as other bones of the face and cranial vault are derivatives of the scaly armor, dermal bone, or exoskeleton which appears first in the early fishes in the scale of evolution.[1] In higher forms this sinks beneath the skin, articulates with the endoskeleton or cartilage preformed bone and forms in the human the bones that ossify in membrane. Even the mandibular articulation is not related to the earlier cartilaginous gill bars but is rather a secondarily formed diarthrosis between two of these membrane bones.

The maxilla ossifies in membrane from three centers on each side, the maxilla proper, the premaxilla, and the prevomer. During its ossification a cartilaginous mass develops in the malar process which, according to Fawcett,[2] probably is either an accessory cartilage or the anterior end of the palato-pterygo-quadrate cartilage. Membranous ossification of the maxilla extends from these three fused centers laterally to include this cartilage, and medially to incorporate part of the lateral wall of the cartilaginous nasal capsule. Thus, while cartilage is present in the field in the embryologic stages, none of the bone of the maxilla appears to be laid down by ossification of it.

The mandible ossifies from one center on either side. This center represents the dentale, a dermal bone present in lower vertebrates. Intramembranous ossification spreads from it to form the body and ramus of the mandible. There are two types of cartilage which develop in the embryonic mandible and become incorporated in the spreading membrane bone. The first is a remnant of Meckel's cartilage. This is an important structure in lower vertebrates. In the newborn infant, however, it is represented only by a small con-

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nective tissue cord, the sphenomandibular ligament, and by a scanty cartilaginous remnant extending along the dental canal to a position just back of and below the incisor teeth. The second type of cartilage to be incorporated into the mandible is the so-called accessory cartilage that develops at the articulation, along the posterior edge of the ramus and the anterior edge of the coronoid process, and at the symphysis. Sections through these cartilaginous areas in the newborn show signs of growth by a somewhat atypical enchondral ossification. Benign tumors that contain cartilage and ossify through cartilage may arise in the jaws. Five such, four benign and one malignant, have been studied by us. They probably originate in connection with remains of these embryonic cartilages, since the relative amount of cartilage present is great.

Repair of fractures and of defects in the mandible and maxilla is by the formation of callus which is fibrous in its first stage and which ossifies usually by direct metaplasia without the appearance of cartilage. However, experi-

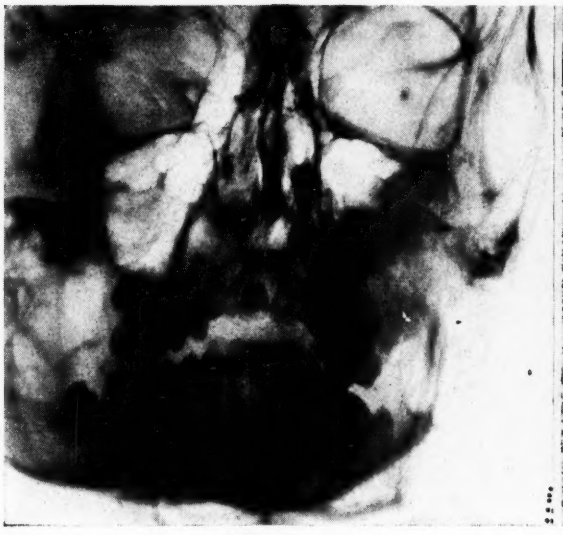

Fig. 1.

Fig. 2.

Fig. 1.—Case 1. Roentgenogram of the tumor of right maxilla.
Fig. 2.—Case 1. Photomicrograph of tissue removed.

ments by Schaffer,[3] Greve[4] and others to be published by one of us (K. S. G.) demonstrate that in the later stages of ossification of the callus and especially when fracture fragments are separated, hyaline cartilage in small amounts may appear and be replaced by new bone by enchondral ossification similar to the callus of a bone preformed in cartilage.

That the great majority of benign ossifying tumors of the jaws are free from cartilage and consist almost entirely of fibrous tissue and bone is to be anticipated from the fact that these bones grow normally by membranous ossification. The findings in the cases to be reported and in those reviewed from the literature substantiate these views. They have been variously designated in the literature as fibrous osteomas or ossifying or osteofibromas, usually according to whether bony or fibrous elements predominate in the particular tumor.

Thirteen cases presenting this lesion, four in the maxilla, eight in the mandible, and one in both bones, have been studied. Two others of the maxilla previously reported by Montgomery[5] are briefly reviewed and illustrations of their pathology included.

### A. FIBROUS OSTEOMAS OF THE MAXILLA

CASE 1.—M. S., white, female, aged fifteen years, was admitted with the history of a growth that had been noticed on the external surface of the right maxilla above and in the region of the first two molars for two years. Examination revealed essentially normal findings with the exception of a swelling of the anterior and anterolateral portion of the right maxilla and a thickening of its entire alveolar margin. Fig. 1 shows the roentgenologic appearance at that time. There is a dense shadow occupying the inferior and lateral half of the right antrum and expanding the walls of the maxilla laterally and inferiorly.

At operation the mucous membrane over the tumor was incised and soft spongy bone comprising the tumor was removed with the exception of portions at the orbital margin,

Fig. 3.

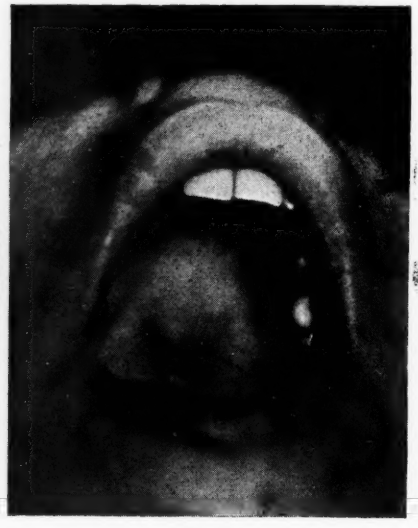

Fig. 4.

Fig. 3.—Case 2. Photomicrograph of tissue removed showing spongy bone, fibrous marrow and giant cells.

Fig. 4.—Case 3. Photograph of the osteoma of hard palate at seat of palatine torus.

about the roots of the teeth, and in the region of the malar bone. The tumor was found to fill about one-half of the region of the antrum. No unossified areas were found.

Microscopic sections (Fig. 2) consisted of trabeculated bone with cancellous spaces filled by immature fibrous tissue. Practically no hemapoietic cells were present. There were rows of osteoblasts along some of the trabeculae. The diagnosis was fibrous osteoma.

The patient received postoperatively 510 roentgen units in divided doses over three months. Six and one-half years later there is no evidence of progression of the tumor and the face is symmetrical.

CASE 2.—W. P., white, male, aged eleven years, had had a gradually increasing enlargement of the anterolateral region of the left maxilla for three years. Examination revealed no abnormalities other than a bony hard tumor in the anterior and lateral portion of the maxilla and including the alveolar process.

The patient was operated upon by Dr. Frederick Moorhead February 4, 1920. A longitudinal incision was made in the mucous membrane of the gum of the left maxilla, and the underlying bony tumor was exposed and chiseled and curetted away. The cavity was packed with gauze.

The specimen consisted of numerous small and large fragments of spongy bone. Microscopic examination (Fig. 3) showed the tissue to be composed of fine bony trabeculae and of a fibrous marrow. The relative amounts of marrow and trabeculae varied in different regions, but nowhere were large islands of purely fibrous tissue seen. Newly forming trabeculae were numerous. Scattered throughout the sections were numerous large foreign body giant cells. There were no evidences of mitosis. The diagnosis was fibrous osteoma.

This tumor is almost identical with that in Case 1 except for the scattered areas containing giant cells. About ten years after operation the growth had remained controlled.

Case 3.—J. McM., white, female, aged twenty-nine years, was admitted with the history that five years before, she first noticed a swelling of the hard palate (Fig. 4). During the last three months the overlying mucosa had become slightly ulcerated. There had been no period of rapid growth.

Examination revealed a bony hard tumor 2 cm. in diameter bulging down from the posterior portion of the middle of the hard palate, a distance of 1 cm., and a small superficial ulcer in the mucosa. A roentgenogram revealed an oval shadow of increased bone density in the region of the swelling.

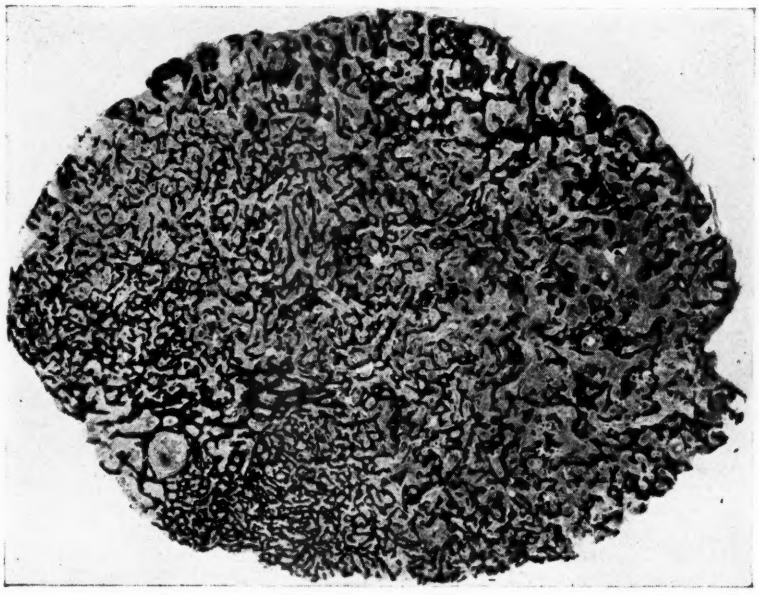

Fig. 5.—Case 4. Photograph of a section at middle of excised tumor.

Under local anesthesia a 2.5 cm. incision was made through the mucosa of the palate, and the oval protruding tumor was chiseled off sufficiently to give the palate its normal contour.

Microscopic examination showed a dense cortical bone with small marrow spaces containing partly fibrosed and partly fatty and hematopoietic marrow. Beneath this cortex was a loose cancellous bone with large irregular marrow spaces that contained a fatty marrow in which a sparse sprinkling of hematopoietic cells was present. This cancellous bone was penetrated by one broad and one narrow band of more compact spongy bone. The trabeculae of the latter stained heavily with hematoxylin and were separated by a richly cellular completely fibrous marrow.

This lesion was at the site of occurrence of the palatine torus and might be regarded as an unusually large hyperostosis, but the presence of much fibrous marrow in the cancellous bone makes it seem more logical to classify it as an osteoma. H. C. Greve[6] is of the opinion that large tori are osteomas.

Case 4.—M. B., white, female, aged twenty-eight years, had noticed a small lump on the maxilla adjacent to the left side of the nose eight years previously. It had enlarged

very slowly. Examination was essentially negative except for the bony hard swelling which bulged forward from the maxilla just beneath the mesial orbital margin and measured about 2.5 cm. in diameter at its base. The Wassermann was positive.

In July, 1913, an oval bony tumor 2 × 2 × 2.5 cm. was removed. Microscopic examination of a section taken through the middle of the entire tumor (Fig. 5) showed it to be made up of fairly dense cancellous bone containing a fibrous marrow which was richly cellular in some regions and markedly collagenous in others. There was a thin irregular cortex along the external surface. There was a very small amount of fatty and hematopoietic marrow


Fig. 6.—Montgomery's Case 2. Roentgenogram of the tumor of maxilla, partly ossified. Unossified portion displaces tooth backward.


Fig. 7.—Montgomery's Case 2. Photograph of tumor removed showing fibrous area partly calcified. *A*, Bone; *B*, calcified area; *C*, fibrous area.

present. The trabeculae in several large areas were more slender, more continuous, and more closely adjacent. There were no areas of active new bone formation and no large areas of fibrous tissue. The marrow spaces were filled with fibrous tissue that was quite mature, with areas in which the cells were separated by numbers of collagen fibers. No areas of fatty or hematopoietic bone marrow were present. Diagnosis: Fibrous osteoma of maxilla.

We have had occasion to study the pathology of Cases 2 and 3, reported by Montgomery, and are reviewing them with the inclusion of illustrations which were not in his publication.

MONTGOMERY'S CASE 2.—A male, aged sixty-six years, had had a tumor removed from the right maxilla twelve years before, but it had recurred in the posterior alveolar region where there was a large oval protruding mass which was bony in its anterior portion and soft posteriorly. A roentgenogram (Fig. 6) shows dense bony shadow in the region of the antrum. At the posterior limits of the maxilla is a shadow of a tooth obliquely placed but with absence of a bony shadow between it and the antrum. Above and anterior to the dense shadow in the region of the antrum is the shadow of a second tooth. There are no shadows of any of the other teeth in the maxilla or the mandible.

The maxilla, including the tumor, was resected. The specimen was sectioned sagittally. Its anterior and superior portions consisted of bone. There was a tooth imbedded in the anterior portion, and in the superior portion there was a yellowish dense calcified area. The posterior and inferior two-fifths, which had grown recently, consisted of soft tumor covered inferiorly and laterally by mucous membrane. There was a tooth imbedded in its posterior portion.

Microscopic examination of the ossified portion showed it to consist of dense cancellous bone. The marrow spaces in the regions that bordered on the soft tumor were filled with

Fig. 8.

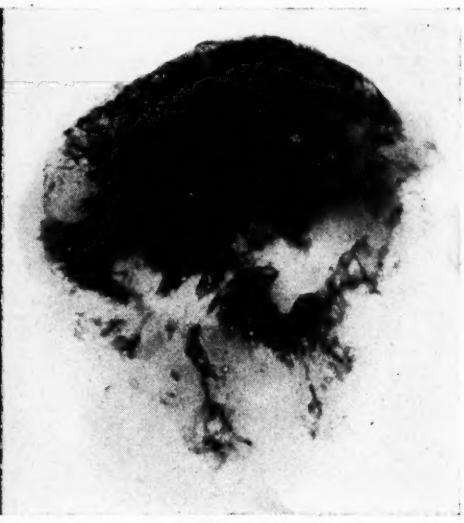

Fig. 9.

Fig. 8.—Montgomery's Case 3. Photograph of buccal surface of tumor removed.
Fig. 9.—Montgomery's Case 3. Roentgenologic appearance of the tumor removed (Fig. 8).

fibrous marrow, while some of those more remotely situated contained fatty and hematopoietic marrow. The dense yellow area in the superior part of the specimen consisted of calcified connective tissue which was undergoing bony replacement at its periphery (Fig. 7). Sections of the soft tumor consisted for the most part of immature connective tissue with wavy collagen fibers and oval to spindle shaped cells. There were mucoid regions in which collagen fibers and cells were few. At the junction of the ossified and unossified portions there were newly formed trabeculae extending into the soft tumor. The diagnosis was ossifying fibroma or fibrous osteoma with areas of calcification and mucoid degeneration.

MONTGOMERY'S CASE 3.—A male, aged sixty-two years, had a hard, inverted, tongue-like projection of eighteen years' duration from the hard palate into the mouth. The tumor was removed. It was reported by Montgomery. We have had occasion to study the pathology of the tumor and are presenting illustrations of it here. Fig. 8 is a photograph and Fig. 9 is a roentgenogram of the excised tumor. More than one-half of the tumor consisted of soft tissue. The superficial portion was fibrous with a covering of mucous membrane, but its deeper portion consisted of bone which extended into the tumor and sprang from the maxilla.

Microscopic examination (Fig. 10) showed that the soft portion consisted of fibrous tissue which in most of its extent was rich in irregularly coursing strands of collagen fibers

and contained a variable number of spindle shaped nuclei. In other places there were large mucoid spaces that were poor in collagen fibers and contained scattered branching and pyramidal nuclei. The base of the tumor contained dense mature bone with fibrous marrow from which irregularly branching rays of bone extended into the soft parts. Diagnosis: Because of the predominance of fibrous tissue in the case the term "ossifying myxofibroma" is more appropriate than fibrous osteoma, although eventually the entire tumor might have ossified.

### B. FIBROUS OSTEOMAS OF MANDIBLE

CASE 5.—A female, aged twenty-nine years, was admitted with the history that at the age of twelve years she had first noticed a tumor of the left side of the body of the mandible. Several teeth were then removed from the involved area. At sixteen years the tumor was partially excised. Sections obtained of the tissue (Fig. 11) showed trabeculated bone with the cancellous spaces filled with richly cellular fibrous marrow. There are scattered giant cells and rows of osteoblasts along the trabeculae.

The tumor had slowly enlarged. Examination at the time of admission was essentially negative except for an oval swelling of the left half of the mandible which extended from

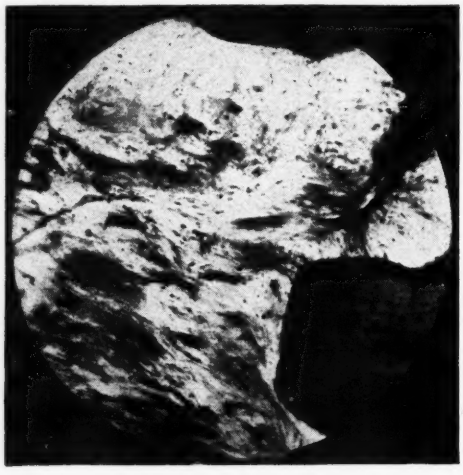

Fig. 10.—Montgomery's Case 3. Photomicrograph of the tissue at junction of bone and soft part of tumor shown in Fig. 8.

the left angle of the jaw forward to the symphysis. There was a loss of sensation in the mucocutaneous distribution of the mental nerve. The mass bulged lingually about 1 cm., buccally about 2.5 cm., and dentally about 0.5 cm. It extended above the level of the remaining mandibular teeth, and in places showed the impressions of the maxillary teeth. Serum calcium and inorganic phosphate were respectively 10.06 and 4.42 mg. per cent.

A roentgenogram (Fig. 12) showed an oval expansion of the bony shadow of the entire left half of the body of the mandible of fairly uniform density. The cortical shadow was greatly thinned. An incision was made along the lateral alveolar margin, and the periosteum and soft parts were reflected downward to expose the bony enlargement. Thirty-five grams of dense spongy tumor bone were chiseled away from the lateral surface to restore the normal external contour of the mandible. The cortex varied in thickness, being very thin in some places. The tumor removed consisted uniformly of very dense spongy bone. No areas of softening were found.

Microscopic sections showed the same type of spongy tumor bone with fibrous marrow, osteoblasts and scattered giant cells as that removed at the first operation. Diagnosis: Fibrous osteoma. The patient made an uneventful recovery. Four months later there had been no recurrence of the swelling.

CASE 6.—A female, white, aged twelve years, was admitted to Presbyterian Hospital with the history that six years previously a bean sized tumor of the gum of the right side of

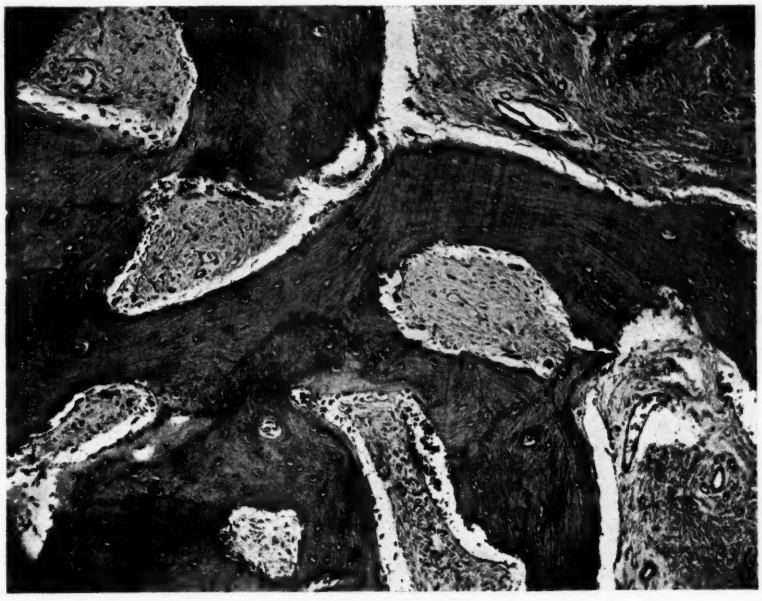

Fig. 11.—Case 5. Photomicrograph of tissue removed.

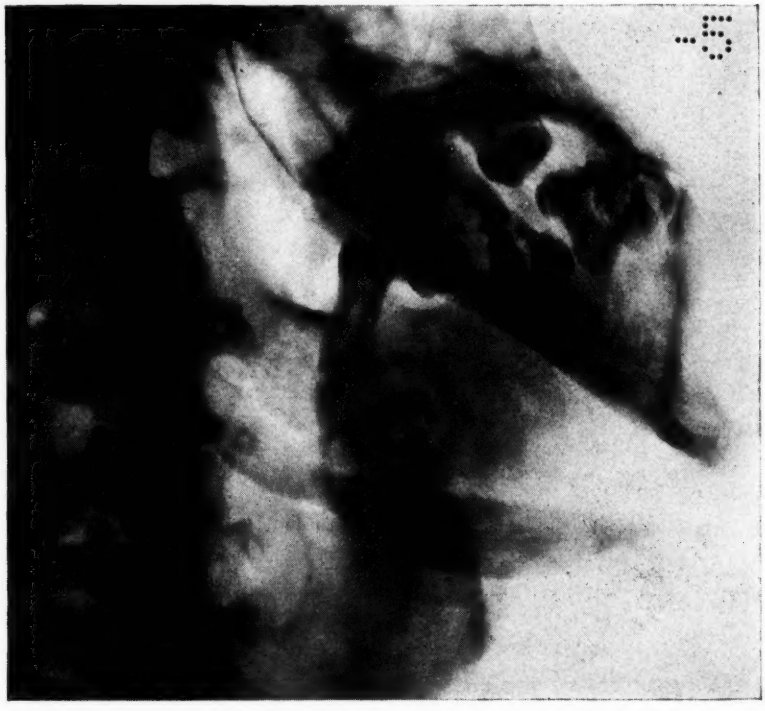

Fig. 12.—Case 5. Roentgenogram of the jaw thirteen years after partial excision of tumor.

the mandible had been excised. It recurred and ten months later had grown to the size of a hen's egg. Treatment with cautery and radium had failed to stop its growth. At the time of admission there was an enormous, firm, oval swelling of almost the entire body of the

mandible on both sides. On the right side all the teeth but the last molar had been extracted, while the five remaining teeth on the left side were markedly displaced.

A roentgenogram (Fig. 13) showed loss of the normal shadow of the body of the mandible except about the left angle. There were radiating streaks of faint bony density extending outward into the faint shadow of the tumor which replaced the body. There were two shadows of radon seeds in its anterior portion.

At operation the entire tumor was removed en masse by Doctor Gatewood. Fig. 14 is a photograph of the superior surface and Fig. 15 of the cut surface of the excised specimen. The tumor consisted of firm, gray, soft tissue containing scattered radiating trabeculae of bone (Fig. 16).

Microscopic examination (Fig. 17) showed it to consist of loose fibrous and myxomatous tissue containing a few radiating spicules of bone. There were no signs of mitosis. Along the periosteal surface, at a point where bony spicules came to the periphery, there was a very thin layer of calcified cartilage which was being replaced by bone and appeared to be formed from the distended periosteum. This was the only region in which cartilage was seen in the tumor. Two explanations for the presence of this cartilage have to be considered. One is that

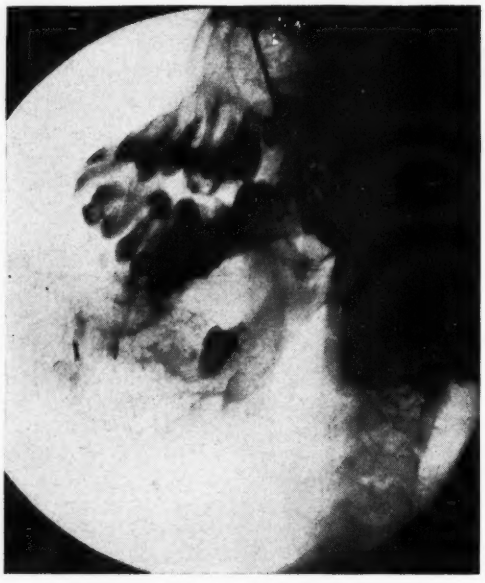

Fig. 13.—Case 6. Roentgenogram of the tumor showing it replacing the mandible and containing faint radiating bone; also two radon seeds.

the tumor arose in bone that was preformed in Meckel's cartilage. By far the more probable explanation is that the tumor arose from membrane, and that in the process of rapid growth a small amount of cartilage appeared in the process of ossification similar to that frequently seen in tumors and proliferative processes in the long bones and occasionally in the healing of fractures of the mandible. Since the fibrous element greatly predominated over the bony, the most appropriate name for the lesion would be ossifying fibroma.

The operative defect was repaired by a curved rib transplant, and the chin was subsequently built up by a series of plastic operations. There has been no recurrence of the tumor in the nine-year interval following the operation. However, the deformity it still severe, and the case illustrates the necessity for avoidance of extensive resection of bone when possible.

CASE 7.—A report of this case has been previously published.[7] A résumé with illustrations is given here.

M. O'N., white, female, aged eighteen years, had a hard oval swelling in the left anterolateral surface of the body of the mandible that had been gradually increasing in size

for four years (Fig. 18). On examination the tumor was of bony hardness. Within the mouth it extended from the second left molar anteriorly and around to the right bicuspid. It also extended upward into the alveolar process about the base of the teeth.

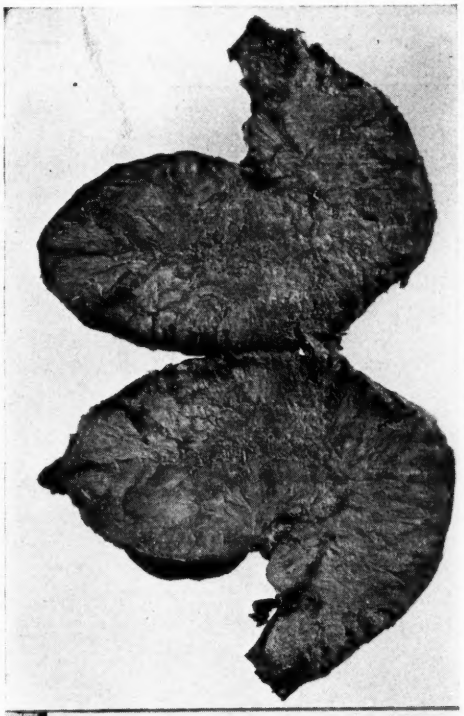

Fig. 15.

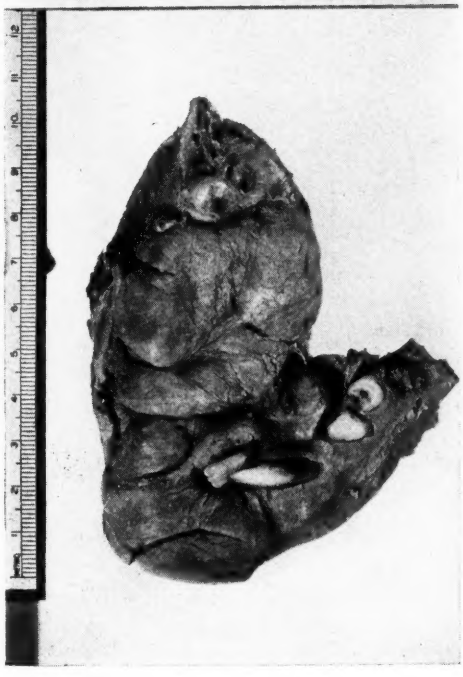

Fig. 14.

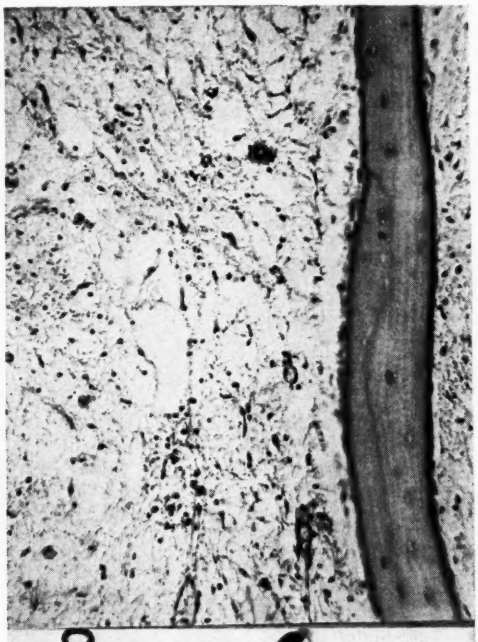

Fig. 17.

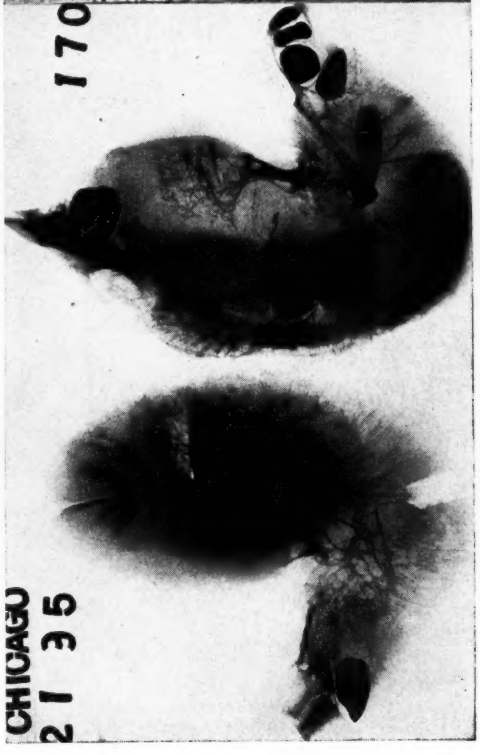


Fig. 16.

Fig. 14.—Case 6. Resected mandible as seen from above.
Fig. 15.—Case 6. Horizontal section of specimen.
Fig. 16.—Case 6. Roentgenogram of bisected specimen.
Fig. 17.—Case 6. Photomicrograph of ray of bone and soft tumor.

A roentgenogram (Fig. 19) revealed the shadow of a large oval swelling of the mandible with a thin dense cortex and a faint mottled interior. The tumor was removed through an intramental incision, and the defect was repaired with a horse-shoe shaped transplant cut

transversely from the upper end of the tibia. On section there was a thin bony cortex and interior filled with firm soft tissue throughout which were scattered small islands of bone.

Microscopic examination showed it to consist of a richly cellular immature fibrous tissue scattered throughout which were islands of spongy new bone. Diagnosis: Ossifying fibroma. Fig. 20 shows the cosmetic result sixteen months afterward. There had been no recurrence of the tumor three years postoperatively.

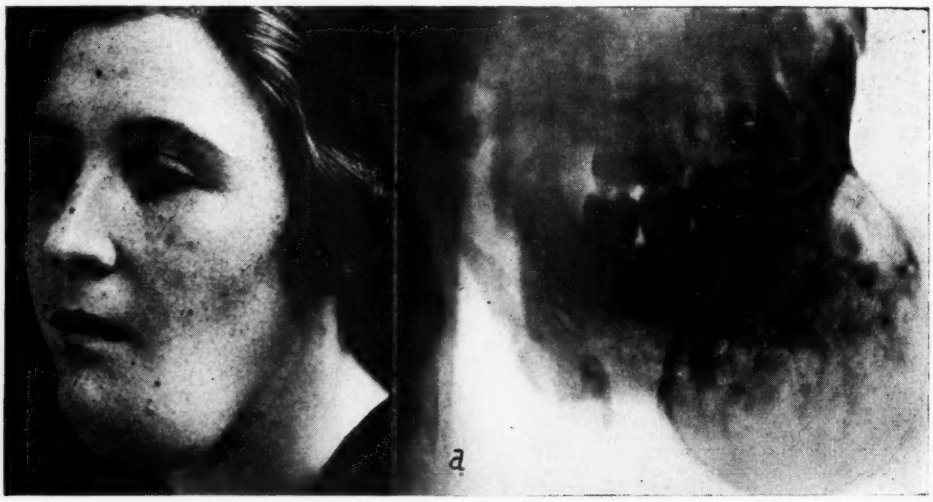


Fig. 18. Fig. 19.

Fig. 18.—Case 7. Photograph of patient preoperatively.
Fig. 19.—Case 7. Roentgenogram of jaw.

Fig. 20.—Case 7. Photograph sixteen months postoperative, showing the cosmetic result obtained by the bone transplant after removal of the tumor.

CASE 8.—E. B., female, white, aged thirty-seven years, had noticed a swelling of the gum of the right side of the mandible eight months before admission, since which time it had very slowly increased. Four months previously a biopsy was taken which showed a mosaic pattern of bony trabeculae and fibrous marrow.

Examination was essentially negative except for a hard, smooth, oval swelling involving both sides of the body and part of the ramus of the mandible on the right side. The body measured 3 cm. in thickness. The teeth were slightly irregularly displaced. Serum calcium and serum inorganic phosphate were respectively 9.89 and 3.34 mg. per cent.

A roentgenogram (Fig. 21) showed diffuse reduction in density with mottling of the shadow of the body and ramus of the right half of the mandible, and a thinning of the shadow of the cortex which was enlarged ovally along the inferior margin.

An incision was made in the buccal mucosa extending from the angle of the jaw forward slightly past the midline. The soft parts were reflected, and a strip of the thin cortex running the entire length was removed. The spongy bone of the interior was curetted out exposing the roots of the teeth in places and damaging the mandibular nerve and artery. The overlying mucosa was sutured and a gauze drain inserted.

Microscopic sections (Fig. 22) showed the tumor to be composed of a mixture of cancellous bone and fibrous tissue. In some places the bone was dense and mature, while in others it was extremely spongy and immature. The unossified portions consisted of spindle cells with bands of collagen fibers in many areas and numerous small vascular spaces. Giant cells were frequent in some places. There was no evidence of cell division. Diagnosis: Fibrous osteoma.

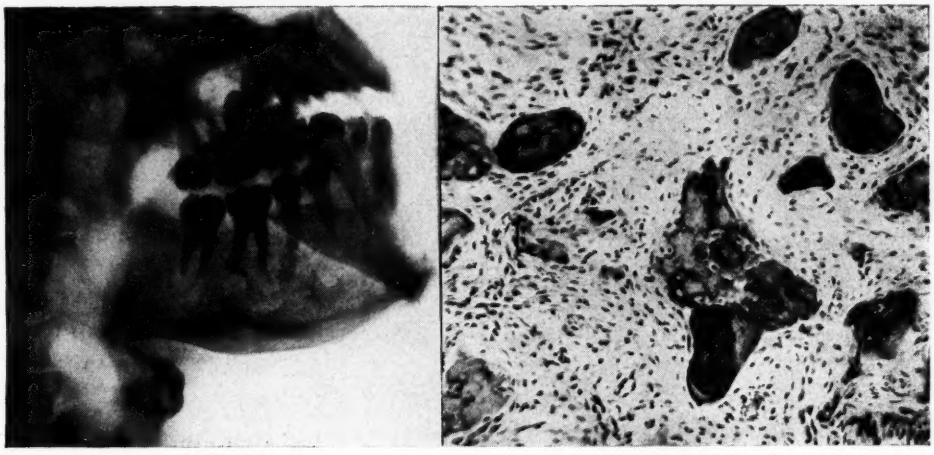

Fig. 21. Fig. 22.

Fig. 21.—Case 8. Roentgenogram of the tumor of body of mandible.
Fig. 22.—Case 8. Photomicrograph of the tissue removed.

The wound healed with little inflammatory reaction. The patient then received 1,465 roentgen units to the region of the right half of the mandible in divided doses. Ten months after operation there was no sign of recurrence of the tumor.

CASE 9.—A. G., female, white, aged forty-one years, was admitted with the history that nine years previously a dentist extracted several teeth and told her that she had a tumor of the mandible. It was then operated upon and subsequently had very slowly enlarged.

Examination at the time of admission was essentially negative except for the right mandible. The molars and second premolars were absent. The right side of the body of the mandible was enlarged from the region of the first premolar backward about 4 cm. Its width was about 2.5 cm. The enlargement involved the inner and outer surfaces of the jaw about equally. Serum calcium and inorganic phosphate determinations were respectively 10.04 and 4.21 mg. per cent.

A roentgenogram (Fig. 23) revealed a large oval area of reduced density in the middle portion of the right body of the mandible with a jagged circular area of greater density in its central portion. No tooth shadows were in the involved region.

At operation an incision was made near the alveolar margin on the buccal and lingual sides of the involved region and enough of the tumor was removed to give the bone its normal contour.

Microscopic examination (Fig. 24) revealed a dense mature bone with fibrous marrow which partitioned irregular islands of partly fibrous and partly ossified tumor tissue. The ground substance of the spongy tumor consisted of an abundance of spindle cells arranged in whorls and bands among various numbers of collagen fibers. Scattered through most of its extent were fine irregular bony trabeculae which stained deeply with hematoxylin. In other regions there were islands of osteoid tissue staining faintly with eosin. Giant cells and hematopoietic cells were absent. Diagnosis: Fibrous osteoma.

The patient recovered uneventfully; 618 roentgen units were then given over the involved area, and five months subsequently there had been no change in size of the bone.

CASE 10.—J. A., female, aged thirty-one years, had had a tumor of the inferior border of the right mandible that had been present for the past eighteen years. Examination was essentially negative with the exception of the scars from an old osteomyelitis of the right tibia and a smooth, hard, small, rounded tumor protruding from the inferior border of the right mandible.

Roentgenologic examination (Fig. 25) revealed a small dense semicircular shadow projecting downward from the lower border of the cortex of the body of the mandible opposite the first molar tooth.

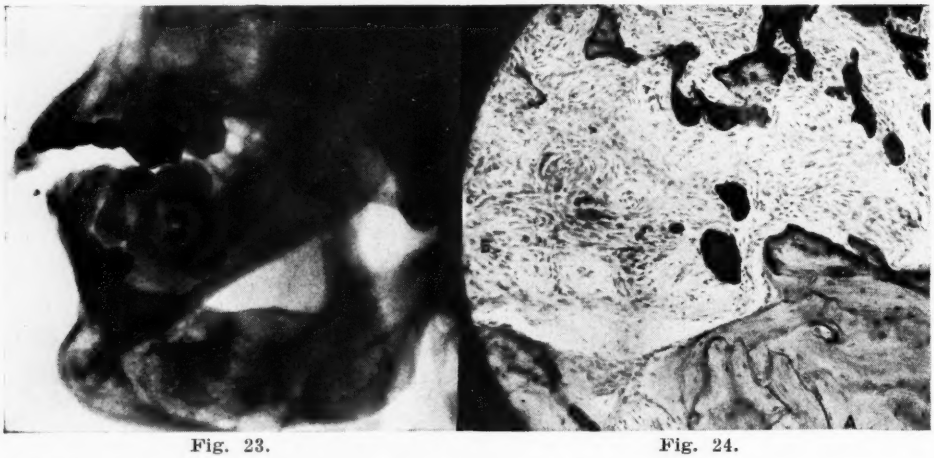

Fig. 23. Fig. 24.

Fig. 23.—Case 9. Roentgenogram of the tumor in body of mandible.
Fig. 24.—Case 9. Photomicrograph showing *A*, dense tumor; *B*, partly ossified tumor.

The tumor was chiseled off under local anesthesia. It was found to consist of a dense bone with no plane of separation between it and the cortex of the mandible.

Microscopic examination of a section through the entire tumor showed a small piece of mandibular cortex included with the base of the tumor. Lamina of mature bone that were continuous with the cortex bulged outward to form the tumor base. The marrow spaces of this cortical bone were small and contained a few spindle cells but no fatty or hematopoietic bone marrow. Peripheral to this was a zone of younger and more irregularly distributed lamina and trabeculae of bone with also small partly fibrous marrow spaces. Between this zone and the cortex was a spongy bone layer with fine trabeculae and large marrow spaces that were partly empty and partly filled with fibrous tissue. The cortex was thin and irregular. In several places osteoclasts were attacking it from the inside. Diagnosis: Fibrous osteoma.

The patient recovered uneventfully and three years later there was no evidence of recurrence.

CASE 11.—This was similar to Case 10 and was associated with a fibrous osteoma of the frontal bone.

J. S., aged nine years, was admitted to the hospital because of a slowly growing bony swelling in the left lateral supraorbital region of two years' duration, which bulged both

anteriorly and into the orbit. Through an incision in the line of the eyebrow the anteriorly protruding portion of tumor was removed. One thousand five hundred roentgen units were given in six doses during the next three months. Six months later there had been no further growth of tumor, but the portion protruding into the orbit was chiseled away because of the deformity which it produced. Three and one-quarter years after the first admission the patient was readmitted because of a pea sized hard bony swelling of three months' duration at the lower margin of the right mandible opposite the bicuspid tooth. There had been no further growth of the supraorbital tumor, although roentgenograms showed slightly increased density of the remaining bone. A roentgenogram showed an oval, even bony shadow protruding from the lower mandibular surface opposite the right bicuspid tooth. At operation six weeks ago it was found to be superficially located and was chiseled off.

Microscopic examination of the half oval shaped section showed it to consist of dense spongy bone with marrow, some of which was fibrous and some fatty and hematopoietic. There was active new bone formation along the surface which was covered by a layer of osteoblasts resembling the cambium layer of the periosteum in infancy.

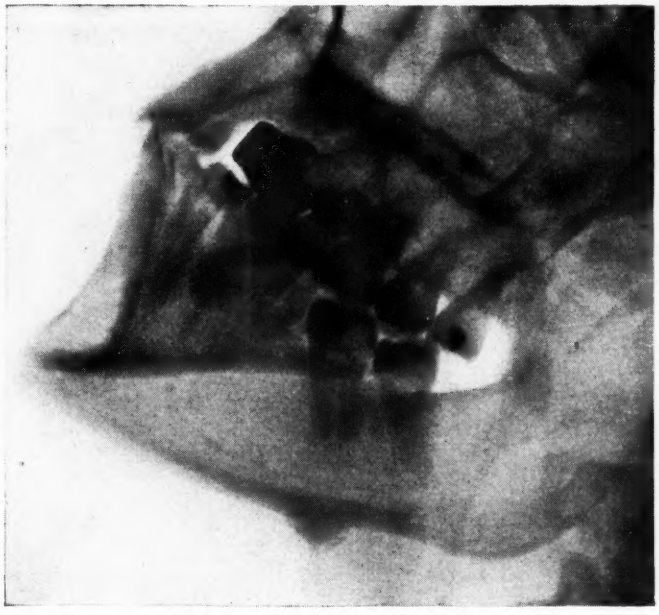

Fig. 25.—Case 10. Roentgenogram of the tumor of lower margin of mandible.

The occurrence of the lesion in association with an osteoma of the frontal bone is good evidence of its benign neoplastic nature and contradicts the view held by some that it is an osteodystrophy or a hypertrophy. Also, the failure of recurrence of tumor after postoperative roentgen therapy of the unremoved portion of frontal osteoma is a point in favor of this procedure for all incompletely removed fibrous osteomas.

Case 12.—C. T., male, aged fifty years, was admitted with the history that six months previously he had noticed a small lump on the lateral side of the alveolar margin just anterior to the angle of the mandible. He believed that it had enlarged slightly.

Examination was irrelevant except for the left body of the mandible. All the teeth had been removed. In the region previously occupied by the last premolar and first molar teeth the alveolar margin and upper half of the body of the mandible were expanded by a hard painless tumor which protruded about 6 mm. buccally and 2 mm. dentally. The overlying mucosa was intact.

A roentgenogram revealed a slight elevation of the alveolar margin of the body of the mandible about 1 cm. forward from its junction with the ramus. The cortical shadow was destroyed. Beneath this in the upper half of the body of the mandible was an irregular

area of reduced density. A small irregular area in its center had a density similar to the uninvolved portion of the body of the mandible.

The tumor was exposed through an incision in the overlying mucous membrane and removed. It involved the entire thickness of the mandible and consisted of a dense spongy bone. The patient made an uneventful recovery.

Microscopically the tumor consisted partly of regions of dense bone with fibrous marrow and partly of regions of spongy bone with a marrow that was partly fibrous and partly hematopoietic. The lesion appeared to be stationary and of long standing. Diagnosis: Fibrous osteoma.

### C. FIBROUS OSTEOMA OF BOTH JAWS

CASE 13.—White, male, aged eight years, entered the hospital with the history of a swelling in the right maxilla noticed by his parents for three and one-half years. A month before his admission three deciduous teeth at the site of the tumor had been removed and a biopsy taken. Examination was irrelevant aside from the jaws. There was a bony hard enlargement of the entire right maxilla which protruded in the infra-orbital alveolar and

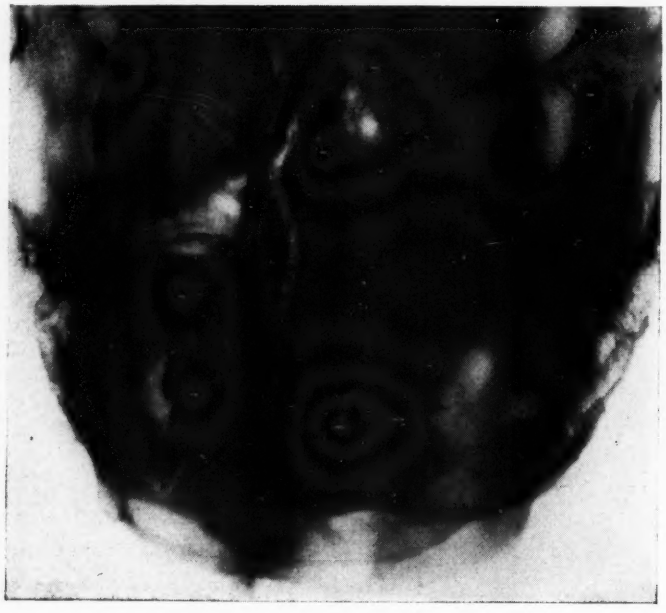

Fig. 26.—Case 13. Roentgenogram showing dense tumor of right maxilla, and a beginning tumor in right side of mandible.

palatal regions. No change was observed in the other maxilla or in the mandible. Roentgenologic examination (Fig. 26) showed a dense radiopaque shadow in the region of the enlarged maxilla and filling out the antrum. There was slight thinning of the shadow of the cortex of the right half of the body of the mandible with slight expansion at the angle of the jaw and an area of circumscribed reduction in density 1 cm. in diameter beneath the permanent premolar tooth.

Under ether anesthesia the mucous membrane along the alveolar margin of the maxilla was incised, and a spongy bony mass was thus exposed involving the whole enlarged maxilla and extending into the border of the malar bone. The wall was cut away and the interior of the maxilla curetted out leaving a thin shell. Three permanent teeth were removed. No antrum was present. There was a spherical mass of soft myxomatous tissue about 1 cm. in diameter in the vicinity of the premolar teeth. The remainder of the tumor consisted of soft spongy bone. No cysts were present. Fig. 27 shows the trabeculae of bone and the fibrous tissue filling the marrow spaces. A section of the soft mass showed it to be composed of myxomatous tissue (Fig. 28). No giant cells or mitotic figures were seen. Diagnosis: Fibrous osteoma of maxilla containing one myxomatous area.

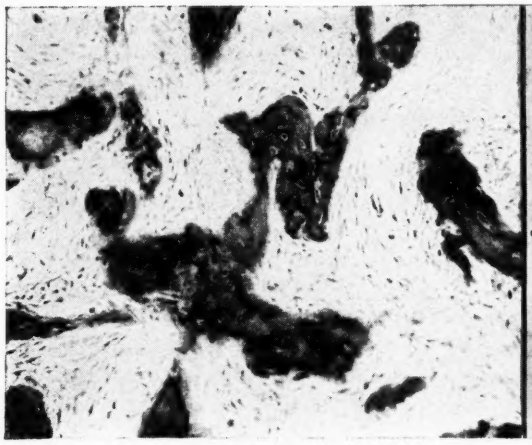

Fig. 27.

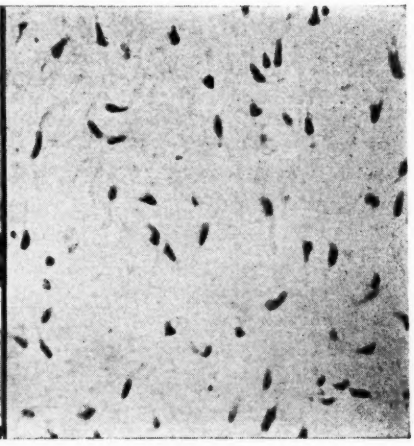

Fig. 28.

Fig. 27.—Case 13. Photomicrograph showing spongy bone with fibrous marrow.
Fig. 28.—Case 13. Photomicrograph showing an area of myxomatous degeneration.

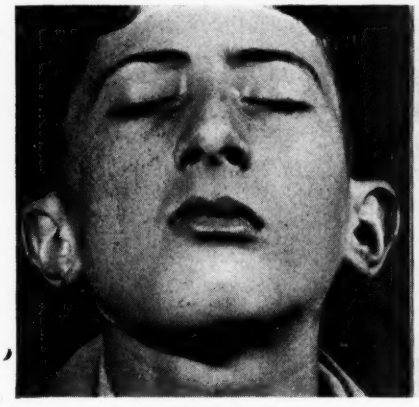

Fig. 29.

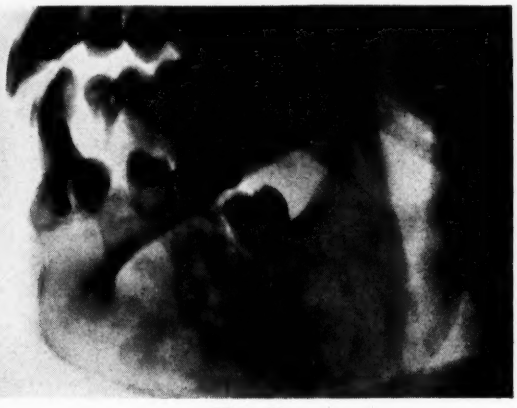

Fig. 30.

Fig. 29.—Case 13. Photograph five years after removal of tumor of right maxilla. The mandible is now the site of tumor formation.
Fig. 30.—Case 13. Roentgenogram made at time of photograph shown in Fig. 29.

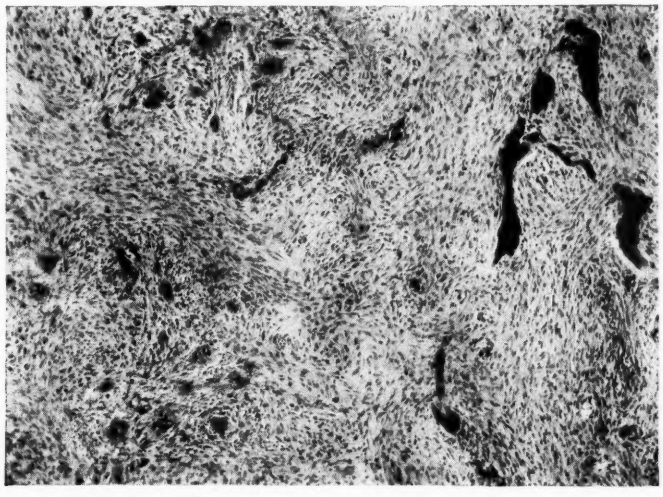

Fig. 31.—Case 13. Photomicrograph of the tissue contained in the tumor of the mandible, showing bony and fibrous areas and many giant cells (×125).

Because some of the tumor about the walls was left behind it was decided to administer roentgen therapy in an endeavor to restrain its further growth. During the next fifteen months the patient received 3,883 roentgen units to the maxilla, and recent examination six years after operation revealed only slight progression in the form of a pea sized nodule on the anterolateral aspect of the maxilla.

A year and one-half after the operation on the maxilla some enlargement was noted of the right half of the body of the mandible. In the next two months it was given 824 roentgen units but the overgrowth continued slowly during the next four years, and it extended into the right ramus slightly past the midline anteriorly (Fig. 29). A roentgenogram (Fig. 30) showed a marked expansion of the shadow of the entire right half and the mental portion of the left half of the mandible. The cortical shadow was thin and the interior faint and mottled with two large areas of greatly reduced density along the inferior margin in the premolar to bicuspid region. Operation was performed for removal of the tumor and restoration of normal contour of the mandible. Serum calcium was 10.08 mg. An incision was made along the inferomesial border of the right half of the mandible and the periosteum reflected to expose the lateral and inferior surfaces of the enlarged bone. Approximately 2.5 cm. of the lateral surface and 2 cm. of the under surface of the mandible were chiseled away. The mental nerve was exposed and preserved. There were two large oval areas of soft tissue which upon microscopic examination proved to be composed of myxomatous tissue of a mature type containing areas of fibrous tissue and areas of calcification and bone formation.

Microscopic examination showed the remainder of the tumor to be more or less extensively ossified. The bone was cancellous and the marrow consisted of cellular fibrous tissue. There were scattered areas of fibrous tissue in which there was no ossification, but in some places there were clusters of giant cells simulating the picture of benign giant cell tumor (Fig. 31).

Thirteen months after the operation there was practically no further growth of the mandible and the facial configuration had become restored almost to normal.

## DISCUSSION

Osteomas of the membrane bones in general are frequent, and the literature, old and new, is replete with cases, most of which are incompletely reported. The majority are tumors of the cranial vault and walls of the accessory nasal sinuses. Carl O. Weber,[8] in 1856, reported that in a total of 95 cases, 43 were of the jaws and 52 of the skull and remaining bones of the face. Sjoberg[9] reports a total of 19 osteomas of the maxillary sinus in the literature up to 1935 and adds two more cases.

It is a common experience of dentists to encounter small osteomas on the lingual surface of the alveolus of the mandibular bicuspid which necessitate removal because of their interference with dentures. According to Partsch[10] this lesion is often symmetrical, and Greve[11] refers to it as symmetrical mandibular tori. Also a small osteoma or hyperostosis, the palatine torus, is not infrequently observed in the posterior midline of the hard palate that is too small to call for surgical removal.

Numerous medical and dental textbooks and journals contain brief and incomplete accounts of cases similar to the 13 here reported. Furedi has recently given incomplete descriptions of 14 cases involving the maxilla. A considerable number of cases have also been reported in detail. They have been variously designated as osteofibroma, ossifying fibroma, osteoma, fibrous osteoma, exostosis, localized osteitis fibrosa, osteodystrophia fibrosa localizata, localized Paget's disease, "intra-osseous epulis," and hypertrophic localized osteitis. A summary is given of 30 detailed reports by Hildebrand,[12] Gangniere,[13] Hippel,[14] Uyeno,[15] Menzel,[16] Mauclaire and Maurel,[17] Monnier,[18]

Montgomery,[19] Moorehead,[20] Kindler,[21] Potts,[22] Konjetzny,[23] Kriegsmann,[24] Renner,[25] Axhausen,[26] and Dechaume.[27] Twenty-four of these tumors began between the ages of eight and thirty-two years. The oldest age of onset was fifty-four years. Only three of the reported cases were followed more than a year. Seven years was the longest postoperative period of observation. Nineteen occurred in either maxilla and seven in the body of either side of the mandible. One reference was found to a tumor of the maxilla and the mandible on one side in the same patient.

No cases were reported to have undergone sarcomatous change. One lentil sized cyst was reported near an antrum in one tumor, and small cyst-like spaces 2 to 5 mm. in diameter were reported in another. Giant cells in small numbers were described in five, and myxomatous areas in one. Trabeculae of new bone in a mosaic pattern and fibrous marrow spaces characterized all the reports.

A history of trauma was emphasized as an etiologic factor in two cases, a history of caries of the teeth and extraction in eleven, and pharyngeal infection in three. Blood calcium had been analyzed in only one case, and it showed a moderate hypocalcemia. A positive statement of the remaining skeleton in ten cases described no other bone pathology.

The first available detailed description of this type of tumor was published by Menzel in 1872. It had been noticed in the mandible at the age of ten and had grown in the next twenty-five years to the size of a fetal head. It was then removed because of threatened obstruction to the air and food passages. The gross and microscopic diagnosis was a benign osteofibroma.

Many years later the histologic resemblance between these localized tumors and the marrow fibrosis with new bone formation of osteitis fibrosa generalizata (von Recklinghausen's disease) was emphasized by numerous surgeons. From this period up to the time of the discovery by Collip,[28] in 1925, of the parathyroid hormone, and the demonstration of its relationship to osteitis fibrosa generalizata by Jaffe, Bodansky, and Blair,[29] in 1930, these jaw tumors were considered by many to be directly related to von Recklinghausen's disease.

In the cases in this study no abnormalities of blood calcium and phosphorus or of the remaining skeleton have been found. Also cysts have been absent and giant cells infrequent. Although the etiology of these tumors is undetermined, there seems to be little indication to relate them to osteitis or osteodystrophia fibrosa generalizata or localizata, and similarly they do not resemble Paget's disease or epulis. It appears that, in a general manner, these tumors have a relationship to membrane preformed bone parallel to the relationship that benign cartilaginous tumors and exostoses have to cartilage preformed bone. This view is substantiated by their tendency to begin in childhood and to grow slowly or not at all in adult life as is the case with cartilaginous exostoses.

A study of the histology of this group of tumors brings out the great variability in the amount of fibrous and osseous tissue. Some tumors are composed of rather mature bone trabeculae with partly fibrosed marrow. Others have islands of fibrous tissue undergoing varying degrees of ossification and calcification. There may also be areas of myxomatous tissue and giant cells.

In a few the tumor is chiefly fibrous tissue with small amounts of ossification. The terms fibrous osteoma, osteofibroma, and ossifying fibroma are often used more or less interchangeably. The more mature tumors with extensive ossification are probably better called fibrous osteomas, while those in which fibrous tissue and immature bone predominate justify the terms osteofibroma or ossifying fibromas. Round cell infiltration and other inflammatory changes were, in general, conspicuous by their absence.

Symmetrical osteomas have rarely been described, most of the symmetrical jaw tumors being reported as fibromas of the gums (Perthes,[30] Rosenstein,[31] Koblin[32]).

## TREATMENT

The treatment of this condition, as detailed in the literature, has been varied. Eleven of the older cases were treated by complete resection, one by partial excision and roentgen therapy, two were biopsied and received roentgen therapy, and three received no treatment after biopsy. The early treatment by massive resection was very disfiguring and carried a high mortality. In view of the benign nature of the lesion it is no longer justified. Biopsy should certainly be performed as soon as the tumor is noticed, since it is, usually, otherwise impossible to establish its benign nature definitely. The decision not to interfere with a tumor may be justified in some instances in view of the very slow rate of growth and slight disfigurement. The majority of authors agree, however, that the tumors should be operatively removed as thoroughly as possible without too great destruction of the jaw bone. It must be emphasized, however, that one operation does not necessarily cure the patient. The condition may recur and require further partial or total resection.

Irradiation has not been generally used in these tumors of the jaw. Six of the cases here reported with incompletely excised tumors received roentgen therapy. One was markedly, and another moderately, benefited, although the process continued slowly in the other jaw despite irradiation, and four were too recently treated to warrant an expression of opinion. In one recent case a frontal bone osteoma that had been partly removed three years previously and then treated by roentgen therapy has been held in check. The experience in treatment of these patients makes it appear that roentgen therapy is beneficial in controlling portions of the tumor not removed at operation. Authors report benefit from radium treatment but also point out the danger of bone necrosis and slough following its use.

## SUMMARY

Thirteen cases of fibrous osteomas of the jaws are reported and the pathology of two other recorded cases discussed. The bone was formed by the process of fibrous or membranous ossification, cartilage being seen in a minute trace in only one case. In twelve cases the tumor consisted largely of cancellous bone and fibrous marrow. In three it consisted largely of fibrous tissue in varying degrees of maturation, and ossification was proceeding slowly.

There was also myxomatous tissue present in three cases and occasional small islands rich in giant cells in two. Microscopic signs of inflammation were rarely present.

## CONCLUSIONS

In general, the tumors are slowly growing, and when starting in childhood tend to become stationary in adult life. No case has been recorded which has become malignant. Blood calcium and phosphorus were determined in four cases and found to be normal. The lesion appears to be a true neoplasm and not a form of osteitis fibrosa, hyperostosis or chronic inflammation. The treatment consists in complete operative removal when the lesion is small and circumscribed. But in cases with diffuse involvement of the bone the operation should, as a rule, be limited to partial removal in order to avoid defects in the jaws and extensive disfigurement. Repeated operations may be necessary. Roentgen therapy was found to retard growth of the unresected portion of tumor for long periods in two cases.

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# USE OF ZINC PEROXIDE IN ORAL SURGERY

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## INTRODUCTION

THE alimentary tract of man and animals normally contains many organisms. These are constantly being introduced with food and any other objects that are put into the mouth. Most of them are killed by the gastric juice, but a few pass this barrier and are carried down through the whole length of the alimentary tract where they may propagate prodigiously. Those which are constantly present and which multiply within the tract may be called normal inhabitants while others are simply transient. Either they are not introduced sufficiently often to be found there, or they do not find the tract a favorable environment for their growth and reproduction. If bacterial cultures are taken from different levels, one finds different species predominant at those levels. For example, the predominating aerobic organism in the mouth is the green streptococcus, and it is found in such numbers when aerobic cultures are made from any part of the oral cavity and is so much more numerous than other organisms that we assume that it multiplies therein and finds an environment peculiarly adapted to its requirements within the mouth. There are, however, a number of other organisms which are frequently found in the mouth and which probably multiply therein but which do not grow easily in ordinary culture media. Probably the first bacteria to be seen by van Leeuwenhoek[1] with his early microscope were of these varieties. They can be seen by an examination of the secretions of the mouth under the microscope either directly or with dark-field illumination, and they can frequently be cultivated on special media or under anaerobic conditions. We do not ordinarily think of them as being present or as being of very much importance in the mouth, but under certain circumstances these organisms play a very important rôle in infections which result from a contamination of the tissues with mouth organisms.

Such infections may occur when the first line of defense has broken down; namely, when there has been a break in the continuity of the surface epithelium due to a wound caused by some sharp object or to the chemical erosion of the surface epithelium or to the direct invasion of the mucous membrane by certain pathogenic organisms. This break in the continuity of the surface epithelium may occur anywhere in the mouth, but by far the most common site is the region of the gums surrounding the teeth, for it is in this location that the mucous membrane is most often injured by the food which is being chewed and by the irritating by-products of food fermentation when food fragments

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have been deposited or packed in between the gum and the teeth or between the teeth themselves. When food is so deposited, it frequently acts as a culture medium for the growth and multiplication of bacteria, unless it is removed before the digestive process has begun to take place. Certainly this happens very often in people who make no attempt to maintain mouth hygiene and in some degree even in those who try to do so. Deposits of hard calcareous substances likewise form on the teeth at the gum level, and as these are built up they frequently press against and erode the surface epithelium of the neighboring gum. When the enamel has been broken or eroded and a cavity develops within the tooth, bacteria may likewise enter the pulp of the tooth and get down into the root canal, or they may pass along the surface of the tooth to the tip of the root.

When a tooth is extracted there is invariably an injury to the surface mucous membrane and likewise a break in the surface of the tooth socket. When fresh wounds are made, whether they be abrasions or lacerations, there is an immediate contamination of their surface with the mouth organisms. Moreover, if a cavity is packed as it frequently is after an extraction, the ordinary drainage and mechanical removal of these organisms fail to take place, and the blood clot and injured tissue which are retained within the tooth socket form an excellent medium for their growth. If these organisms are present in small numbers, the inoculation is relatively small; and, if the neighboring tissues are relatively normal, there may be little danger of their gaining a foothold; but in most cases in which an extraction is done the neighboring tissues are already diseased, the organisms are already present in large numbers and have developed an ability to invade these tissues. Further injury to the tissues renders likely deeper invasion. It is true that in long standing cases there may be a local barrier which has developed in the tissues, and if the tooth is removed with a minimum of injury to this barrier it may still be able to resist the invasion of the organisms. If, however, there is a recent acute inflammation with swelling, redness, pain and fever, we must assume that the organisms have greatly increased in number and have already penetrated whatever protective barrier may have been built up. In such a situation if a tooth is removed even with a minimum of injury, the likelihood of deeper penetration of the infection is very great.

For reasons which we have mentioned above, two things stand out as of great importance: first, minimizing the injury to the tissues, and, second, minimizing the number of contaminating organisms. Experienced dentists understand the importance of the first of these, and those who are bacteriologically minded understand the second.

It is with the hope that the second of these important factors may be materially improved that I venture to apply to these dental infections, experience which I have gained in my study of surgical bacteriology.[2-6] This field of medicine is in fact the borderline between dentistry and surgery, and such problems can best be worked out by the dentists and the surgeons working in cooperation. This is a particularly important field, because when an infection spreads from the oral cavity to the tissues of the face or neck and it is necessary to em-

ploy surgery to halt the progress of the infection there is always the necessity for disfigurement by the production of a visible scar and in many cases a threat to life itself by the rapid spread of the infection beyond the reach of surgery either into the brain or into the mediastinum.

## ANATOMICAL FEATURES

To understand the spread of infection from the region of the teeth to other tissues, a knowledge of the anatomy is essential. Infection may spread by three routes: first, by means of the lymph channels; second, by means of the blood vessels; and, third, by means of direct extension. Coller and Yglesias[7, 8] have recently employed these points.

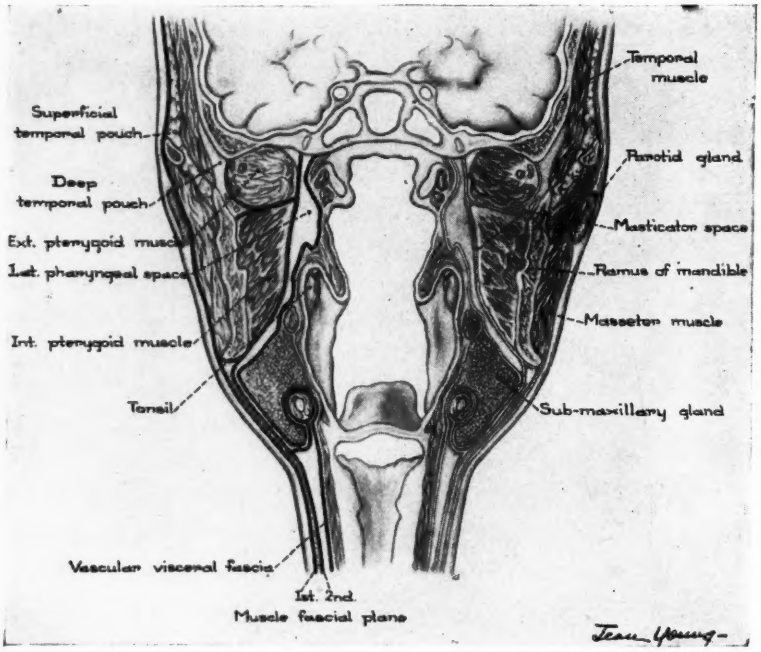


Fig. 1.—Frontal section demonstrating the different fascial planes and potential anatomical spaces. (From Coller and Yglesias, Surg. Gynec. Obst., 1935.)

The lymphatics of the oral cavity drain into the collar of lymph glands below the jaw.[9] The central portion of the chin, the lower lip, the lower gums and the tip of the tongue drain downward into the submental lymph glands, but there are communications also with the more lateral lymph glands which lie in close contact with and occasionally in the substance of the submaxillary salivary glands in the submaxillary triangles, which take the main drainage from the cuspids, bicuspids, and first and second molar teeth. The region of the third molar may also drain into the submaxillary lymph glands, but the drainage frequently passes backward to the deeper glands at the upper part of the jugular chain. The pillars of the fauces and tonsils as well as the palate generally drain to the upper jugular lymph glands, but there are communications from those regions also which pass down into the internal maxillary nodes and retropharyngeal nodes. The tongue lymphatics may drain into the lingual nodes

or may pass directly into submaxillary nodes or into the upper or lower jugular glands, the principal node of the tongue lying at the bifurcation of the common carotid artery. The region of the teeth in the upper jaw generally drains to the submaxillary nodes on either side.

The blood vessels of the oral cavity also serve as a route by which infection may spread, chiefly of course along the venous branches which follow well-known anatomical distributions. Most of the drainage is downward through the anterior facial, ranine, lingual and pharyngeal veins into the internal jugular and through the superficial temporal, internal maxillary, and posterior facial into the external jugular, with free communications between these two systems and with the smaller anterior facials in front. But of particular importance are

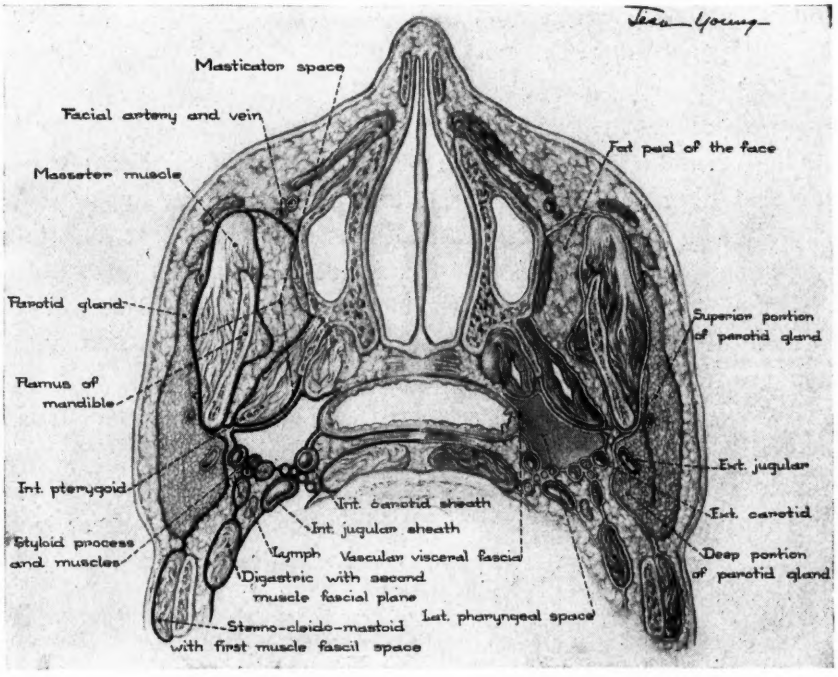


Fig. 2.—Horizontal section demonstrating the different fascial planes and potential anatomical spaces. (From Coller and Yglesias, Surg. Gynec. Obst., 1935.)

communications between these vessels, in which there are no valves, and the ophthalmic veins, so that if there is any blockage to the downward flow of blood, it may flow back through the pterygoid plexus into the inferior ophthalmic and through the angular vein at the inner angle of the eye into the superior ophthalmic, and thus into the cavernous sinus. Not infrequently when infections involve the upper jaw these veins become thrombosed and the clot is propagated up into the cavernous sinus, and when infection spreads within the clot or along the wall of the vein, infection enters the skull and death ensues.

The fascial spaces of the face and neck also direct or limit the spread of infection. Some of these fascial spaces contain muscles to which the fascia is firmly adherent. Others contain glands, others contain bone, and still others contain blood vessels.

The superficial cervical muscular fascia which includes between its two layers the sternocleidomastoid and the trapezius muscles passes upward to the lower border of the mandible and then fuses with the periosteum on the anterior surface of the mandible in front, but posteriorly it passes over the surface of the masseter muscle and under the parotid gland. The second cervical muscular fascial layer includes the sternohyoid and omohyoid muscles of the neck and passes up and fuses with the superficial layer above the hyoid bone, but separates from it again to pass posterior to the mandible and then continues upward to surround the external and internal pterygoid muscles. This fascia tends to limit infections which occur in the body of the mandible, but in the posterior portion around the third molar tooth infection more readily spreads upward, in what has been called the masticator space, including the ramus of the mandible and the masseter and pterygoid muscles. This space then continues upward where infection may spread either superficial to or deep to the temporal muscle.

The third layer of cervical muscular fascia encloses the sternothyroid and thyroid-hyoid muscles and continues on above the hyoid bone to cover the mylohyoid and digastric muscles. This fascia passes under the submaxillary gland and posteriorly it fuses with the fascial sheath of the internal jugular vein. Within these muscular fascial spaces lies a fascial cylinder which extends from the base of the skull and the floor of the mouth down to the fibrous portion of the pericardium. This is called the viscerovascular layer and contains the pharynx and esophagus, larynx and trachea, the thyroid and thymus glands, as well as the big vessels, nerves and lymphatics and their surrounding fat. It is divided into compartments, but infection spreads easily upward or downward when it has once entered this cylinder, and thus infections which enter the floor of the mouth may very rapidly spread downward to block off the larynx by the accompanying edema or pass downward into the mediastinum.

## PATHOGENESIS OF MOUTH INFECTIONS

Anaerobic and microaerophilic organisms play a prominent part in the inflammatory processes in the gums as in cases of pyorrhea, or in the ulcerated infections of the tongue and cheek, commonly called stomatitis. Whether they are primary invaders of normal tissue like the hemolytic streptococcus or the diphtheria bacillus is not known, but they frequently invade the tissues when there has been a break in the primary defenses of the mucous membrane, either caused by a foreign body such as a splinter or a fish bone or a tooth, or following an operative procedure such as tonsillectomy or tooth extraction. When such an invasion takes place, they may simply produce a diffuse inflammation of the submucous tissues or they may produce necrotic ulcers which destroy large portions of the mucous membrane, or they may spread through the lymphatics to the cervical lymph glands, or they may spread by direct extension through the tissues causing extensive necrosis either downward through the floor of the mouth and into the neck or upward into the face, not infrequently extending up into the orbit or beneath the temporal muscle into the pericranial tissues.

If the infection manifests itself as a severe cellulitis of the submucous tissues, either of the mouth or of the throat, it gives the typical picture described by Plaut[10] and by Vincent,[11] commonly known as Vincent's angina. If they invade the floor of the mouth and get down into the tissues of the neck or into the lymph glands of the neck, they may threaten or produce a closure of the glottis which is called Ludwig's[12] angina.

### DIFFERENTIAL DIAGNOSIS

Mild forms of inflammation of the mucous membrane of the mouth occur in patients after prolonged vomiting or prolonged starvation. The digestive action of gastric juice may produce ulcers in the mouth or on the lips which do not persist long after the vomiting ceases. In starvation cases the stomatitis has been attributed to lack of vitamins, and this is confirmed by the favorable response following the administration of these substances in many cases. Smears and cultures from these lesions usually do not reveal the organisms mentioned above in number indicating activity.

The hemolytic streptococcus commonly produces a diffuse cellulitis of the tissues of the throat and not infrequently spreads to the neck, and by the rapid development of edema frequently threatens or produces a closure of the glottis, but there is usually no necrosis of the mucous membrane or of the deep tissues of the neck, and the odor of the exudate is not foul, while infections in which the anaerobic or microaerophilic organisms are playing the dominant rôle, result in both necrosis of the mucous membrane and necrosis of the deeper tissues and the exudate has a rather characteristic and offensive putrid odor. Usually the hemolytic streptococcus infections produce a high fever and a profound intoxication, while the infections due to the anaerobic and microaerophilic organisms produce less fever and less intoxication and a more distressing and extensive destruction of tissue. The hemolytic streptococcus infections are very apt to arise acutely and to subside rapidly, while the microaerophilic and anaerobic infections are more apt to develop slowly, to spread insidiously, and to resolve less rapidly.

Surface cultures in the hemolytic streptococcus infections reveal large numbers of those organisms. The ordinary aerobic cultures in the other type of infection usually yield only the ordinary mouth organisms with the green nonhemolytic streptococcus predominating. However, if a dark-field illumination is used for examination of the exudate, fusiform bacilli, spirilla, or spirochetes may be seen. Both *Treponema macrodentium* and *T. microdentium* are found.[13] The spirochetes apparently are almost always present in the severest cases, but may be absent from the mildest forms. When anaerobic cultures are made, nonhemolytic streptococci or microaerophilic streptococci are usually found on the blood agar plates, and with the special media recommended by Smith and others, the fusiform bacilli, spirilla and spirochetes may be obtained. The fusiform bacilli grow out in forty-eight hours in plain cooked meat medium to which 10 per cent ascitic fluid has been added. The spirochetes grow out in eight to ten days in 33 per cent sheep or beef serum in distilled water to which a bit of

sterile rabbit kidney has been added. These organisms are not pathogenic when injected into animals in pure culture, but when they are combined they have a synergistic action which produces a lesion more or less extensive.

The odor of the lesion arises both from the development of gases by the bacterial action on the tissues and from the necrosis of the tissue itself. When these organisms are cultivated in media, it is found that the nonhemolytic anaerobic streptococcus is the only one which in pure culture will produce a foul odor. The spirochete *T. microdentium* produces an unpleasant odor but it is not foul. When the streptococcus and the spirochetes are combined, the odor is very foul and resembles that coming from the infected tissues.

One must of course be on the lookout for a combination of these two infections, in which both the aerobic hemolytic streptococcus and microaerophilic or anaerobic organisms are found. It is curious, however, that the infections arising in the region of the tonsils are almost always due purely to the hemolytic streptococcus, and those arising from the teeth are almost all due to the anaerobic organisms.

## TREATMENT

When anaerobic organisms are present in the mouth unaccompanied by any inflammation, they may be minimized or eliminated by a number of oxydizing agents such as potassium chlorate, potassium permanganate, or sodium perborate, but they are more quickly and more effectively eliminated by the use of zinc peroxide. When, however, there is an infection either mild or grave with an invasion of the submucous tissues, and superficial ulceration of the mucous membrane, zinc peroxide is much more effective in treatment than the other oxydizing substances. When the infection has spread to the deeper layers either directly or through the lymphatics, surgery is required and should be radical, extending well beyond the areas of indurated tissue and opening the involved fascial spaces. It is frequently necessary to remove masses of lymph glands or the submaxillary salivary glands in order to effect the proper drainage. If the incisions have not been carried to the limit of the necrosis, it will continue to spread and cause a great deal more destruction. This has been illustrated over and over again when conservative operations have been attempted in the dental clinic, for example, and the patients have subsequently had to be taken into the hospital for more extensive surgery. When the tissues have been opened up by adequate surgery, however, the infection can be brought under control by the careful application of zinc peroxide suspended in sterile distilled water, to every part of the infected surface. If this is done the foul odor will almost immediately disappear, and smears and cultures of the exudate will show rapidly decreasing numbers of the spirochetes, fusiform bacilli and the anaerobic streptococci.

The zinc peroxide must be an effective preparation as shown by preliminary tests indicating its ability to liberate oxygen when suspended in distilled water. Only the "medicinal grade" should be used. The preparation now supplied by the du Pont Chemical Company, Niagara Falls, New York, is effective. It should be sterilized in small quantities at 140° C. dry heat for four hours, and applied as follows. The dry powder is suspended in approximately an equal

quantity of water so that it has the consistency of 40 per cent cream. It can be mixed nicely with an asepto syringe and applied with the syringe to every part of the wound surface. If there are any sinuses, it should be delivered into them with a catheter; but, if there are any parts of the infection which cannot be reached, the wound will have to be opened so as to permit contact, which is essential. When the whole surface of the wound has been covered, fine meshed gauze soaked in zinc peroxide should be placed over the surface, and the whole wound sealed with several layers of vaseline gauze to prevent evaporation. The dressing should be changed daily. When the gauze is removed at the end of twenty-four hours, the old zinc peroxide and exudate should be washed off with saline from an irrigating can. The zinc peroxide suspension is then applied as before.

The zinc peroxide should also be used at the same time as a mouth wash, about one part powder to four parts water. One mouthful every three to four hours coats the mucous membrane very well. This should be thoroughly spread around the surface of the mouth. Good contact can be assured by applying the medicine to the gums or any other affected areas by means of cotton swabs. If there are any lesions on the tonsillar walls or pharynx, these should be sprayed with the suspension. Gargling or swishing the material around in the mouth will usually not result in contact with the tissues back of the anterior pillar of the fauces.

When the smears and cultures have become entirely negative for pathogenic organisms, simple dressings will be satisfactory. Our experience with these cases during the past two years has demonstrated the superiority of this treatment over any previous treatment, first, in the rapid subsidence of acute general symptoms, second, in the rapid resolution of the local process, third, in the rapid disappearance of the foul odor which is so offensive to the patient and to other patients in the neighborhood, and, fourth, in the rapid healing of the wound.

I believe that zinc peroxide should be used as a prophylactic mouth wash in all cases preliminary to tonsillectomy or dental extraction. It is logical to believe that these serious infections would be greatly diminished if this were used extensively. Preliminary studies of the flora of the mouth should demonstrate the presence of the offending organisms; and, if they are present, treatment should be instituted a day or two before the operative procedure is carried out, until the organisms have largely or, if there is time, until they have completely disappeared.

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# LUDWIG'S ANGINA

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## INTRODUCTION

WHAT has made Ludwig's angina or angina Ludovici so important is the frightful rapidity and certainty with which an unchecked case proceeds to a fatal termination. It is proved, by the recorded cases, that it is more rapidly fatal than similar infections in other parts of the neck than the submaxillary region. Its high mortality justifies us in believing that it is an infection of a dangerous character. The prognosis of this disease is very uncertain in that it may take the life of the patient within twelve or fourteen hours, or it may begin and continue as a mild affection for several days and suddenly assume an alarming character.

## DEFINITION

Ashhurst[1] defines Ludwig's angina as "a clinical entity, consisting of a septic cellulitis of the floor of the mouth and of the neck." According to Brophy,[2] "Ludwig's angina is an acute infection of the floor of the mouth usually starting in the region of the submaxillary salivary glands. The infection may involve the glands, but it is usually below them and very deep seated." Hayes[3] states that "Ludwig's angina is an acute phlegmonous inflammation of the tissues of the floor of the mouth and sides of the neck." Keen[4] defines Ludwig's angina as "an acute inflammation of the lymph nodes and connective tissues within the capsule of the submaxillary salivary gland." Stengel[5] states that "Ludwig's angina is a septic inflammation of the tissues of the floor of the mouth surrounding the submaxillary gland." Thomas[6] made the following statement: "A cellulitis localized to the submaxillary region, regardless of the kind of infection, in the writer's opinion, is not a Ludwig's angina but becomes one as soon as the process invades the floor of the mouth and the pharynx." MacFarland[7] states "the infections that occur in the neighborhood of the submaxillary glands are not always confined to the glands themselves, but may rapidly extend to the surrounding cellular tissue, with resulting phlegmonous and even gangrenous changes. Such cases are described as angina Ludovici, or Ludwig's angina, and can scarcely be regarded as a disease of the salivary glands, but rather as a form of cervical cellulitis." Von Wagener and Costello:[8] "The disease which has been so set apart under Ludwig's name has been defined with unmistakable clearness in Foster's *Encyclopaedic Medical Dictionary* as 'a diffuse phlegmonous inflam-

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mation of the floor of the mouth and the intermuscular and subcutaneous tissue of the submaxillary region which may end in gangrene, abscess or resolution and which sometimes prevails in an epidemic'."

The definitions all agree that it is a cellulitis of the floor of the mouth above the mylohyoid muscles, but may show submandibular involvement.

## HISTORY AND NOMENCLATURE

Parker in 1879 published a historical review of this condition. Auretius gives details of cases which seem to be those of Ludwig's angina, but he called the condition "cynanche." Paulus Aegineta spoke of a similar condition which he called "paracynanche." Dr. Fothergill gave an account of "Putrid Sore Throat" (1739-46) which appeared to have some of the characteristics of Ludwig's angina. Dr. Kirkland in 1786, Dr. Wells in 1809, and others reported cases of this type.

In 1836, Ludwig presented the first accurate description of this dangerous condition, which he called "gangrenous induration of the neck." Cameron, in the following year, named it Ludwig's angina.[6]

Von Thaden, in 1872, published reports of eighteen cases; he regarded Ludwig's angina as an acute bubo of the lymph glands below the angle of the jaw, and advised that the name be given up. Twenty cases were added in 1883 by Roser. The cervical fascia received much attention from Koenig (1882) and from Paulsen of Copenhagen (1886). The latter added a wealth of statistics from an analysis of the reports of 251 cases of submaxillary phlegmon, in twenty-two of which the floor of the mouth was invaded.[9]

The French surgical society[6] in 1892 showed great interest. Members divided into two parties, one being led by Nélaton, the other by Delorme. Nélaton took the stand that Ludwig's angina should not be recognized as a separate disease and was instrumental in having resolutions to this effect passed by the society. At the following meeting Delorme caused this action to be reconsidered and Ludwig's angina to be given its proper place in surgical pathology. In 1893 Leterrier wrote a thesis, the chief object of which appeared to be to support the position of his teacher, Delorme, who contended that Ludwig's angina was primarily a sublingual phlegmon.

In 1895, Semon's paper appeared, in which he maintained that acute septic inflammation of the throat and Ludwig's angina were pathologically identical, thus eliminating Ludwig's angina as a separate disease.

It has been suggested by Ludwig, Tissier, Roser and Chalin that it is a morbid entity. "Submaxillary bubo" was the term used by Thadden. Chantemesse considered it a true erysipelas of the larynx. Delorme called it a subgingival phlegmon.

## ETIOLOGY

Generally speaking, the primary cause of this infection is carious teeth or diseased tonsils. Ashhurst[1] makes the statement that the predominating rôle of dental infection is the cause in 65 to 83 per cent of all cases. Hayes[3] does not limit the cause to abscessed teeth, but also includes osteomyelitis,

septic needles or solution in anesthetizing the mandible for surgical procedure or traumatism. Houser,[10] MacNevin and Vaughan,[11] Muckleston,[9] MacFarland[7] and Wassmund[12] attribute the cause to infected posterior teeth of the mandible. According to Casselberry[13] and Sautter[14] this condition has its origin in the tonsils and peritonsillar abscesses. MacCallum[15] reports a case in which the infection was thought to be caused by an infected abrasion of the skin of the neck. Stengel[5] states that Ludwig's angina "may result from carious processes at the roots of the teeth, or from infection of the submaxillary lymphatic glands in the course of various infectious disease, particularly scarlet fever." A medicolegal[16] case was reported, and the following explanation was given for the accident following extraction: "The infection was caused either by the entrance of germs on the dental instruments used or from cotton used by the dentist in his treatment or by some other means; but at all events germs found their way into the tooth socket, did their deadly work and produced an unusual, unlooked for and unforeseen result—death."

In the literature on Ludwig's angina no specific organism is mentioned. Von Wagener and Costello[8] state that streptococci are present in primary Ludwig's angina, while staphylococci are found in secondary Ludwig's angina. Thoma[17] mentions that this virulent infection is caused by hemolytic streptococcus generally derived from odontogenic or tonsillar infection. Spencer[18] relates a case in which cultures were taken from the wound in the neck. *Streptococcus pyogenes* was the main organism found, both in cover-glass specimens and upon cultivation. The cultivated throat cultures also showed a yeast staphylococcus albus and a few bacilli.

### CLINICAL PATHOLOGY

The most frequent termination is abscess formation (this occurs in the majority of cases) with perforation externally or into the mouth. Occasionally, however, a rapid necrosis or gangrene results. The problem that is most obscure in Ludwig's angina is the cause of death, whether it results from septic intoxication or from invasion of the air passages. If death is due to septic intoxication, then this condition is to be explained by the presence of a rare and especially virulent infection. If, on the other hand, invasion of the respiratory tract is responsible, then the mortality is to be explained by extension of the phlegmonous inflammation to the larynx and in some cases to the lungs. An understanding as to how this disease progresses may best be obtained by the knowledge of the anatomy of the oral cavity. Thomas[19] gives a very detailed description: "The muscular floor of the mouth is formed by the two mylohyoid muscles which fuse with each other at the anterior median raphe. This muscular diaphragm separating the mouth from the neck is a complete one from the posterior edge of one mylohyoid muscle to that of the other and is a comparatively strong one. There are no openings in it for the passage of planes of connective tissue between the mouth and the neck. From the posterior border of the mylohyoid on each side, extend backward the constrictor muscles of the pharynx separating the pharynx from the neck,

the muscles of the two sides fusing together at the posterior median raphe. The three constrictors, superior, middle and inferior, overlap each other, so that here also, the submucous tissue of the pharynx is not continuous with the connective tissue of the neck through these muscles. Between the posterior edge of the mylohyoid and the anterior border of the middle constrictor, however, is a considerable deficiency in the buccopharyngeal muscular wall. This opening extends from the hyoid bone upward and backward to the inner side of the lower jaw near its angle. The hyoglossus muscle, which, viewed externally, forms a part of the floor of the submaxillary triangle, does not enter into the formation of the floor of the mouth or pharyngeal wall. It passes upward through this muscular opening or gap to become a part of the root of the tongue and fills the gap considerably. These structures which pass from the neck into the mouth or in the opposite direction, do so through this opening. These are the glossopharyngeal and hypoglossal nerves, the lingual artery and vein, and the styloglossus muscle. The greater part of the opening, however, is occupied by the deep portion of the submaxillary salivary gland which here projects into the floor of the mouth, near the root of the tongue, where it may, therefore, be said to form a small part of the floor of the mouth. The submaxillary gland within the mouth is adjacent to the posterior part of the sublingual gland and is attached to it by the surrounding loose connective tissue. We thus see that the connective tissue in the submaxillary fossa is directly continuous with that in the floor of the mouth, so that the extension of a submaxillary cellulitis to the sublingual region, which occurs so early and so constant in Ludwig's angina, is readily understood.

"The mouth with the jaws closed may be roughly compared to a small box of which one side has been removed. The upper side or roof is represented by the roof of the mouth, the lower side or floor by the two mylohyoid muscles, the front and lateral sides by the teeth and jaws. The posterior side is absent. With the jaws closed the mouth is practically filled by the tongue and the normal sublingual tissue. Therefore, when the cellular tissue under the tongue is invaded by inflammation, as in Ludwig's angina, the tongue is pushed upward and the mouth must open to make room for the new inflammatory material. Speech and deglutition are necessarily interfered with and the saliva now increased by the inflammation cannot be properly swallowed and frequently escapes from the mouth. The tongue crowded for room may show between the teeth and appear to be swollen when it is not. Posteriorly the tongue becomes wider and dips downward and backward toward the larynx, where the base of the epiglottis is attached to its posterior surface. Laterally the base of the tongue reaches the sides of the pharynx, where it receives the attachments of the styloglossus and palatoglossus muscles. These attachments of the sides of the tongue to the walls of the pharynx, make on each side a strong muscular ridge covered by mucous membrane and submucous tissue, the latter being scanty here. This prominent ridge separates the floor of the mouth from the pharynx, so that a submaxillary infection extending through the opening already described, and finding itself in the floor of the mouth in front of this ridge, must extend through it along the intermuscular fascia or over it along the scanty submucous tissue. This ex-

plains why the swelling in the floor of the mouth is so well developed, before the edema has produced alarming symptoms in the pharynx and larynx. The finger placed in the mouth will easily find this ridge. Since the tongue turns downward and backward, the sublingual swelling lies in front of this posterior portion, so that the tongue with the epiglottis attached to its dorsum is pushed backward toward or against the posterior wall of the pharynx, tending to obstruct the air which is passing from the nose and mouth to the lungs. By the same mechanism in anesthesia, the dropping backward of the tongue and epiglottis may interfere with respiration. The submaxillary salivary gland lying in the opening in the floor of the mouth is about on a transverse plane with the base of the tongue, i.e., just anterior to the larynx; so that the portion of the gland projecting into the mouth is only about two inches external and anterior to the larynx. The chief protection of the larynx, at first, is the muscular ridge already described."

## VARIETIES

Von Wagener and Costello[8] believe that Ludwig's angina is of two varieties—a primary Ludwig's angina and a secondary Ludwig's angina. The differences between the primary and the secondary types are as follows: First, in primary Ludwig's angina the immediate source of focus of infection from which the sublingual tissues become involved is in the cellular tissue around the lymph nodes. Usually in secondary Ludwig's angina there is an infection of an abscess of the lymph nodes themselves in the mental, submental, or anterior submaxillary regions.

Second, in primary Ludwig's angina the infection spreads through the loose areolar tissue about the posterior edge of the mylohyoid muscle. In the secondary the path of spread is a direct one through the muscles or through their median raphe.

Third, the sublingual phlegmon in secondary Ludwig's angina represents, as a rule, the same types of a cellulitis found about an infected lymph node or the periphery of an abscess in any locality.

## SYMPTOMATOLOGY

Keen,[4] Blair and Ivy,[20] Prinz and Greenbaum,[21] Muckleston[9] and Ochsner[22] agree on the symptoms of Ludwig's angina. Chills and high fever characterize the inception of the disease, followed soon by difficulty in swallowing and breathing. The outstanding symptom in Ludwig's angina is a rapidly spreading, hard, boardlike swelling beneath the border of the mandible on one side, which may in the course of twenty-four to forty-eight hours involve both sides and even the front of the neck. The patient complains of pain in the floor of the mouth, stiffness in movements of the tongue, pain in efforts to clear the throat, and increased salivation, the saliva being of a fetid odor. The edematous floor of the mouth is pushed up to a level with the tops of the teeth and is covered with a grayish slough. The mouth is held open to allow room for the protruding tongue, and, as a consequence, swallowing and respiration are greatly embarrassed. If untreated, symptoms of grave sepsis

develop: and if the patient survives long enough, there will be discoloration of the skin with diffuse suppuration or partial gangrene of the deeper tissues. If no relief is obtained, the patient becomes cyanotic and may die in from seven to twenty days from suffocation combined with toxemia from the absorption of inflammatory products.

### DIAGNOSIS

The following points Ludwig considers to be diagnostic:[19]

First, "the insignificant inflammation of the throat, which often disappears entirely after the first few days, and which if it persists may be looked upon as superficial."

Second, "the 'woodlike' hardness of the swelling, which does not pit on pressure."

Third, "the hard sublingual swelling, forming a ring just within the lower jaw, reddish or bluish in color."

Fourth, "the sharp limitation of the indurated tissues which are surrounded by uninvolved healthy connective tissue. The slight involvement or more often lack of involvement of the glands although the inflammation attacks the connective tissue around the glands."

### PROGNOSIS

The prognosis of a sublingual phlegmon will vary according to the virulence of the infection. Septic intoxication, which occurs in Ludwig's angina, varies according to the virulence. The prognosis depends upon the promptness with which the condition is recognized and upon the thoroughness of treatment. Thomas[19] reports that in a considerable number of cases although free incision was made no pus was found and in these death usually resulted. Where pus was found early, a cure usually followed; although in some, other foci existed and a fatal termination was the result.

Muckleston[9] in his study of Ludwig's angina states that Thomas' compilation remains the fullest in statistical summary. In 106 cases reported in the literature and met with in his own practice, there was a fatal ending in forty-three, a little over 40 per cent. Yerger collected, from records of the Cook County Hospital, twenty-three cases with a mortality of 43 per cent. Coakley states that "several cases of this suppurative type are seen each year in our service at the Bellevue Hospital, and all have recovered after free external incision into the abscess."

### TREATMENT

Blair and Ivy,[20] Prinz and Greenbaum,[21] von Wagener and Costello,[8] and Brown[23] advocate immediate and free incision of the area with the aid of a local anesthetic. The surgical procedure suggested by Blair and Ivy[20] is used universally by the other authorities. The skin is infiltrated with a ½ per cent solution of novocaine, and an incision is carried from the tip of the chin to the hyoid bone and from the latter point outward under the angle of the jaw as far as the outer margin of the induration. The median incision and

the median part of the lateral incision are carried boldly through the deep fascia, but as part of the lateral incision might pass over the facial vein and carotid arteries it is made with more precision. A flap is drawn upward, exposing the digastric muscle and the lower part of the submaxillary gland. If there is any induration in the floor of the mouth above the mylohyoid, this muscle and the anterior belly of the digastric are cut. If the induration has crossed the midline, the same incisions may be made on the other side, but in no instances are the geniohyoid muscles to be cut. These incisions allow the indurated sections to roll outward, which frees the floor of the mouth. If the induration has extended down the neck below the hyoid bone, a vertical incision is made in the midline, which allows two more triangular flaps to be retracted. The wounds are packed with gauze and never sutured. If, after proper incisions in the floor of the mouth, restlessness persists and is not relieved by a sedative but is partially relieved by allowing the patient to sit up, then tracheotomy is to be considered; and, if one can be satisfied that the respiratory impediment is not due to pneumonia, then tracheotomy should not be delayed too long.

Lindsay[24] suggests that it is essential that the general toxemia should be treated early by salines and saline purgatives, and the patient's general condition supported throughout the course of the disease. The hygiene of the mouth should be regularly attended to by swabbing and irrigation. The local condition should be treated with fomentations in the early stages. Should the cellulitis be progressing, multiple incisions should be made to lessen the pressure from edema and to improve the local circulation. If possible, this should be done under local anesthesia, as, owing to the presence of edema of the fauces, the administration of a general anesthetic is attended with considerable risk. If an anesthetic has to be given, chloroform alone should be used.

Brown[23] suggests the following treatment for the general health of the patient. The patient must be kept in bed and fed on a nutritious, light, liquid diet; though, if the dysphagia is severe, it may be necessary to rely on nutrient enemas. In the early stage the bowels should be opened with calomel. A mixture containing strychnine and the perchloride of iron and of mercury should be given. Antistreptococci (antiscarlatina) serum (20 c.c. or even more) should be given as soon as possible and repeated next day if necessary, the usual precautions as to anaphylaxis being observed. Local treatment to the throat consists in giving small pieces of ice to suck, spraying with warm alkaline lotions, and in cases of laryngeal edema the frequent exhibition of 1:5,000 adrenalin, alone or combined with 5 per cent cocaine.

Levy[25] reports a case which he treated with acriflavin. Twenty c.c. of a 1 per cent solution of neutral acriflavin per injection was introduced intravenously on three successive days. Eight days after the onset the patient was discharged completely well.

Houser[10] states that it is remarkable, however, that more attention has not been given to the relief of this condition by intraoral operation early in its onset, before much cervical involvement has occurred. In the large series of cases reported by Thomas, there were seventeen of spontaneous evacuation, all the abscesses opened internally; in one there was both an internal and

an external opening. If the patient is seen early, there is a possibility of bringing the infection to a speedy termination with relatively simple intraoral surgical maneuvers. One advantage of this operation is its simplicity, with the result that it will often be applied more quickly than the more commonly used external operation, which often is properly delayed in hope that spontaneous subsidence will occur. No anesthesia is required, and the associated pain is probably not so great as that experienced in the opening of an abscess of quinsy. If the condition is so far advanced that the mouth cannot be opened, the external drainage route is the only one possible.

## CASE REPORT

This case has been taken from the records of the Windber Hospital, Windber, Pennsylvania, and clearly illustrates the rapidity of this disease. The patient was an Italian miner, aged thirty-seven years. He was admitted to the hospital with the chief complaint of difficulty in breathing and swallowing, redness and swelling of the jaw and neck. He had always been in good general health, and there was no available history of any chronic disease in the family. On Tuesday, July 24, 1934, he went to work in the mine. The following morning (Wednesday) he complained that a tooth in the lower left jaw was sore, but there was no swelling. On Thursday morning (July 26) the left side of his jaw was swollen, with evidence of dysphagia. The swelling continued, and by evening there was great swelling of the mandible and the soft tissues of the neck. By this time dyspnea and dysphagia were very pronounced, although he had little or no pain. The swollen tissues were red at this time. He was admitted to the hospital at 9:30 P.M., July 26, 1934. The physical examination showed a patient well developed and well nourished, but apparently acutely ill. He was mildly cyanotic and had very marked dyspnea. There was a phlegmon of the subglossal space extending to the submental region and down the neck. There was a great deal of swelling and edema producing pressure on the pharynx and larynx. The tongue was inflamed and swollen, and the gums were infected. Dental hygiene was poor, and the tonsils were swollen and acutely inflamed. Temperature 102° F.

Progress notes are as follows: July 26, put to bed. Patient complained of not being able to swallow. Expectorated much mucin. Croup kettle was used continuously. To have each hour a 1:5,000 potassium permanganate gargle followed by normal saline solution. July 27, gargle as ordered. Restless at times. Croup kettle used continuously. Noticeably worse at 5:45 P.M. Attending physician notified and patient removed to the dispensary and had tracheotomy performed. Patient seemed improved after operation. Pulse weaker at 9 P.M., cyanotic. Respiration ceased at 9:22 P.M.

## CONCLUSIONS

The primary cause is frequently local infection of the mouth or trauma, caries being the most common finding.

The opening in the muscular buccopharyngeal wall, through which the submaxillary salivary gland projects into the floor of the mouth, is the path by which the submaxillary infection invades the mouth and pharynx.

Streptococci are always present and sometimes staphylococci are found.

The pathologic lesion is that of a cellulitis, spreading from the tissues around the salivary glands to the opposite side of the throat and to the neck.

Abscess formation occurs in a great many of the cases.

The first symptoms are dysphagia and dyspnea, later toxemia.

Intravenous therapy has been found to be of advantage in some cases. Early intraoral drainage may bring the infection to a speedy termination. Either an intraoral or an external incision will relieve pressure caused by the edema and cause the patient to be more restful.

Pharyngeal abscesses are indicative of cervical suppuration; their safest approach is by an external incision. The wound should be packed with gauze in cases of severe hemorrhage. If respiration becomes too difficult and labored, a tracheotomy may be the last resort.

From this study it is evident that Ludwig's angina is a very dangerous infection, in that one never knows with what rapidity it may progress. Early treatment is to be encouraged, and the patient should be under constant observation because of the high mortality rate of the disease.

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**The Rôle of Nutrition as a Factor in Resistance to Dental Caries.** May Mellanby, Brit. D. J. **62**: 241, 1937.

The differences between dental clinicians and research workers are partly due to the different angles from which they approach the problem in question. The clinician often fails to appreciate the value of an adequately controlled investigation, while the scientist believes that clinical observation, in order to be of value, must be able to withstand criticism successfully.

We know today that dental caries is rife in the civilized world not because of the absence or presence of any one factor. It is not caused by eating excess sugar, by not cleaning the teeth, by eating soft food, by omitting to chew detergent food at the end of our meals, by neglecting to use this or that tooth paste. Many factors play a part; some of which are known, some unknown.

Nevertheless, experimental and clinical investigation has shown that the deficiency of fat soluble vitamins, especially vitamin D, is the main single factor in temperate zones; at the same time, the presence of sufficient vitamin D is responsible for the comparative freedom from the disease of the unspoilt natives of arctic and tropical climes. One must, of course, also appreciate that the type of food may influence the reaction of the saliva and so indirectly affect, from the outside, the development of caries. Furthermore, the direct physical or chemical effect of food upon the surface of the teeth has also an influence on the occurrence of caries, which may be termed the influence of diet; while nutrition deals with the food after it has passed from the mouth into the alimentary canal and is digested. Animal experiments show conclusively how the tooth structure may be altered by varying certain factors in the diet during the period of tooth development (Fig. 1).

Some years back only 3 vitamins were known: A, the fat soluble growth vitamin; B, the water soluble growth vitamin; and C, the antiscorbutic vitamin. Gradually, evidence accumulated which showed that there were two fat soluble vitamins, both of which were promoting growth, but one had to do with epithelial tissues and infection, the other with calcification. Unfortunately, the term vitamin D was introduced to describe one-half of the original vitamin A complex instead of using terms similar to those employed for the complex vitamin $B_1$, $B_2$, etc.

Factors assisting calcification are present in egg yolk, milk, animal fat, cod liver oil, and irradiated ergosterol. The amount varies in different samples of the same food, particularly in milk, depending upon the food eaten by the animals and the amount of sunlight to which animals and foods are exposed, for the ultraviolet rays of the sun acting on the food or on the unprotected skin synthesizes vitamin D.

In order to grade the structure of human teeth, a collection of deciduous teeth was examined. About 80 per cent of the teeth showed a greater or lesser degree of imperfection or hypoplasia, but only 3 per cent showed

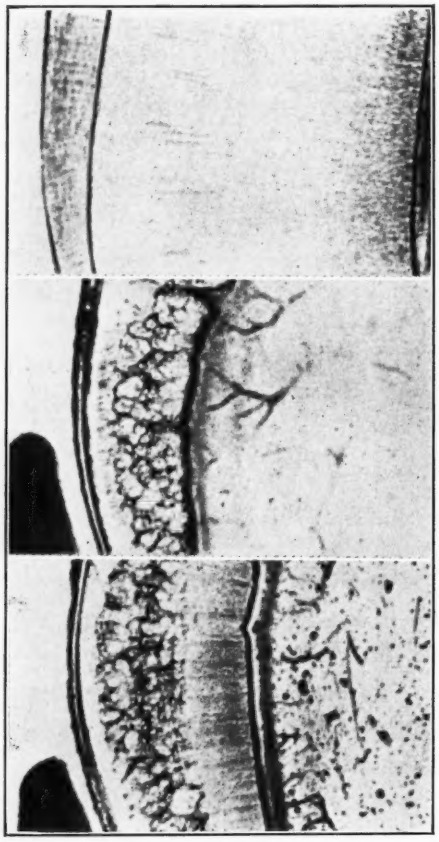


Fig. 1.—Photomicrographs of sections of the permanent teeth of three puppies from the same litter. The basal diet was the same for each: *A*, was given ample fat-soluble vitamins. Note thick enamel and dentin; *B*, diet very deficient in fat-soluble vitamins. Note very thin enamel and dentin. The enamel is pigmented and the dentin contains numerous interglobular spaces and large uncalcified areas; *C*, for the greater part of the period of growth the diet was the same as for *B*; note the structures are similar. For the last four weeks much fat-soluble vitamins were given and a thick band of well-calcified dentin was laid down. The original badly calcified tissues were, however, unaltered. Period of experiment: *A* and *B*, four months; *C*, five months.

typical textbook hypoplasia (Fig. 2). When the teeth were examined for surface defects and later on sectioned histologically, a definite association between the two findings became apparent. Therefore, it is possible to predict roughly from a surface examination of a deciduous tooth what its histologic structure will be. To grade the surface enamel, a probe is rubbed lightly over the teeth, particularly over the buccal surface. Then the surface is examined

by oblique lighting. Similar methods of examination can be used for judging the surface structure of permanent teeth. The correlation between surface structure and caries is shown by the following data:

Of 1,500 sections examined, there was no caries in

78 per cent of normal teeth
46 per cent of slightly hypoplastic teeth
8 per cent of moderately hypoplastic teeth
6 per cent of very hypoplastic teeth

Excessive caries occurred in

7.5 per cent of normal teeth
13 per cent of slightly hypoplastic teeth
46 per cent of moderately hypoplaste teeth
74 per cent of very hypoplastic teeth

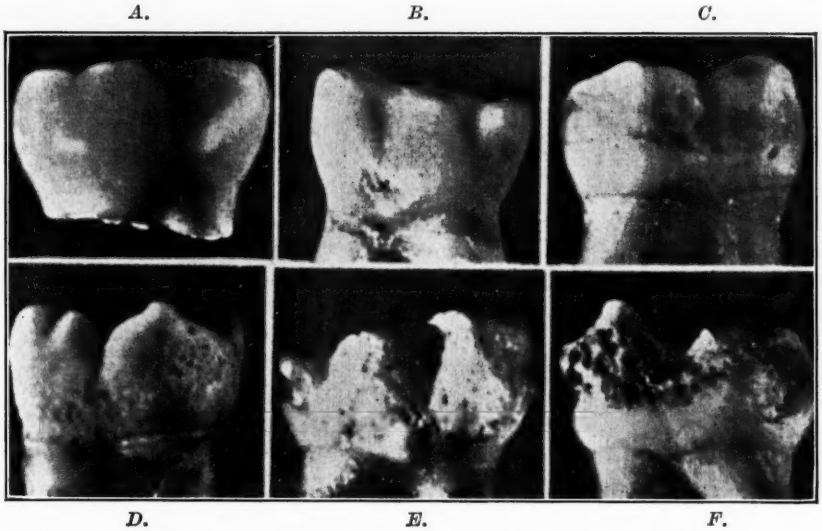


Fig. 2.—Photographs of a series of second deciduous molars to show some grades of hypoplasia compared with the normal. *A*, Nearly normal; *B*, slight hypoplasia; *C*, hypoplasia; *D*, severe hypoplasia; *E*, severe hypoplasia; *F*, "gross" hypoplasia.

The dentin of hypoplastic teeth also shows signs of disturbance; it is thin and contains many interglobular spaces. Even the formation of secondary dentin is influenced by the vitamin D contents of food. If artificial injury (rubbing with a file) is produced on a puppy's tooth, the secondary dentin formed depends on the diet eaten prior to and during the period of injury. If the diet is rich in calcifying properties, the secondary dentin is well formed and abundant; if the diet is insufficient in vitamin D, there is but little dentin laid down and it now contains many imperfectly calcified areas.

From these investigations it seems possible that the character of the secondary dentin gives an indication of the power of resistance at the time of injury. Furthermore, when studying the relation of caries to the structure of the tooth, a few teeth of good structure were found to be decayed, while a few of bad structure were free from disease. Upon examination, the secondary dentin was well calcified when caries was not found in hypoplastic teeth,

while it was badly calcified when caries was found in teeth with a perfect enamel surface. This indicated that a change in the diet had occurred from the time the enamel was formed until the period when the secondary dentin was laid down. This suggested that by giving children a good calcifying diet the progress of caries may be delayed. A nutritional investigation of this type was undertaken with children in a hospital for surgical tuberculosis. The results are summarized in Table I.

TABLE I

THE SPREAD AND ARREST OF CARIES IN CHILDREN'S TEETH
(Average Age of Children, Nearly Six Years)

| DIET | DIET VARIATIONS | AVERAGE PER CHILD | |
|---|---|---|---|
| | | TEETH SHOWING INCREASE OF CARIES | "DEGREE" OF ARREST OF CARIES |
| 1 | Least fat-soluble vitamins | 5.0 | 0.2 |
| 2 | Little extra fat-soluble vitamins | 3.3 | 1.2 |
| 3 | Much extra fat-soluble vitamins | 1.4 | 3.7 |
| 4 | Irradiated ergosterol (vitamin D) | 1.0 | 3.9 |
| 5 | Irradiated ergosterol (cereal-free) | 0.4 | 4.7 |

Later on, investigations on similar lines with more children who were in an average state of health were conducted. The results corroborated the early findings.

These facts also fit in with the geographical distributions of dental caries. The latter is very prevalent in countries such as Britain where cereals are cheap and largely eaten, and where foods rich in vitamin D (egg yolk, milk and fat) are expensive. There is little sunshine, and neither bodies nor foods are fully exposed to it. On the other hand, cereals are rarely eaten by the Eskimos, whose diet is rich in animal fat containing vitamin D. The diet in tropical countries includes cereals and is not rich in vitamin D, but the skin of the inhabitants is abundantly exposed to the sun, especially in childhood; in this manner, the necessary vitamin D is synthesized in their bodies. Breast feeding is prolonged both in the arctic and the tropical zones. As soon as civilization reaches these people, conditions alter in relation not only to diet but also to clothing.

In order to reduce caries in temperate zones, radical changes are necessary in diets and habits. The consumption of milk, eggs, cheese, animal fats and vegetables must be increased and that of cereals diminished. Breast feeding must be general and prolonged. Cod liver oil or some other form of fat soluble vitamin should be given to all infants from birth.

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## Local Anesthesia

**The Clinical Foundation for Local Anesthesia of the Face, Jaws, and Oral Cavity.** (Die Klinischen Grundlagen der Localanästhesie des Mund-Kiefer-Gesichtsbereiches.) August Lindemann, Deutsch. Zahn-Mund-Kiederhlk. **2**: 400, 1935.

The Westdeutsche Kieferklinik in Düsseldorf for a number of years has investigated the causes of after effects of a general nature occurring during the use of local anesthesia. They found that none of the newer drugs can displace novocaine. It is important that the solution, however, is isotonic, of body temperature, and of the same reaction as the body tissues. They prefer normosal (Sächsische Serumwerke, Dresden) as solvent. To produce an isotonic 1 per cent solution use 0.75 gram normosal to 100 c.c., for a 2 per cent solution 0.5 gram normosal is added. Normosal contains sodium, potassium, calcium, magnesium, bicarbonate, phosphate, and chloride.

For vasoconstrictor action suprarenin is still used extensively, but clinical experience has shown that corbasil eliminates many of the untoward effects of the former drug, especially cardiac complaints, increase in blood pressure, and suprarenin shock. It is admitted, however, that these are caused mostly by too high dosage, by faulty technique of injection, and too rapid resorption (introduction into a vessel). For five years the synthetic product prostigmin was used. Local anemia is not so marked when using corbasil or prostigmin as when using suprarenin. While this seems a disadvantage to some operators, it is considered an advantage by the author, as it protects the tissue from infection, makes for the formation of a healthy blood clot, and quick healing without secondary hemorrhages or the formation of dry sockets in cases of extraction. He advises the operator to select for each case the anesthetic solution that will give the best results rather than using the same routine combination. He also gives a general procedure which according to his experience is important for successful performance of oral surgical operations with local anesthesia: The evening before the operation, or in emergency cases one hour before, 0.5 gram of veronal is given by mouth. A short period before the operation is started an intramuscular injection of 2.2 to 3.3 c.c. of pernocton is administered to adults. This dosage is not sufficient to cause narcosis or sleep, but is used for its general quieting effect on the patient.

The anesthetics which have been found best for the various methods are as follows:

For surface anesthesia the use of anesthetizing salves is advised, especially pantokain. When administered into the nares, it causes anesthesia of the incisors.

For local injections novocaine plus corbasil is advised.

For conduction anesthesia the author recommends novocaine plus prostigmin. It is important, however, to inject very near the nerve; endoneural injection is recommended.

**Roentgen and Clinical Investigation of the Spreading of the Anesthetic Solution in Intraoral Conduction Anesthesia.** (Röntgenologische und Klinische Untersuchungen über die Ausbreitung des Anesthetikums bei intraoraler Leitungsanesthesie.) Adolf Isaak, Schweiz. Z. Zahnhlk. **45**: 955, 1935.

To determine the spreading of the anesthetic solution injections were made by the use of neo-iodipin-panthesin-adrenalin, which is radiopaque and therefore serves to determine to what extent the tissue becomes infiltrated. The roentgen pictures showed that the anesthetic solution spreads extensively and therefore produces extensive local anesthesia in the area of injection.

To determine the extent of the action of the anesthetic solution the limit of the anesthetized area was determined clinically. The following are the results:

1. Injection at the infraorbital foramen causes complete anesthesia of the region of the canine. The distal part of the second incisor is affected, but posteriorly the effect decreases; at the first premolar it is 80 per cent, the second premolars 40 to 32 per cent, the first molar 19 to 8 per cent. Medially the median line is reached in only 14 per cent of the cases.

2. The zygomatic injection produces total anesthesia on the buccal side of the tuberosity, and the area of the third and second molars. Anterior to this the effect decreases. In only 4 per cent is the first premolar anesthetized.

3. The mandibular injection produces anesthesia on the lingual side of the alveolus as far as the first premolar in all cases, and in 90 per cent it reaches as far as the cuspid, in 71 per cent as far as the second, and in 48 per cent as far as the first incisor. On the buccal side the area of the third molar is affected in 83 per cent of the cases. The area of the second molar to the first premolar is not affected, but both distal and mesial to the cuspid anesthesia will result, but it decreases toward the first incisor on account of the nerve supply from the other side. [The writer says on account of nerve fibers anastomosing, but nerves do not anastomose.—Reviewer.]

4. The buccinator injection (made medially to the anterior margin of the coronoid process at the level of the maxillary occlusal plane) causes the best anesthesia on the buccal mucosa of the first molar, the distal half of the second premolar and the mesial half of the second molar.

**Methods of Anesthesia for Operations in the Surgical University Clinic of Heidelberg.** (Die Art der Schmerzausschaltung bei Operationen in der Chirurgischen Universitäts Klinik, Heidelberg.) D. Philippides, Chirurg. **8**: 13, 1936.

The percentage of operations performed under local and general anesthesia has increased the last years in favor of local anesthesia.

Ethyl chloride was used in 1 per cent of the cases, especially for minor operations, and for children, except infants.

Ether, generally induced with ethyl chloride was used in 6 per cent of adult patients, and 28 per cent of children. Often local anesthesia is used in combination to prevent undue bleeding and to decrease the amount of ether used.

Chloroform was not used except for children for pylorus spasmus operations.

Nitrous oxide was used in 2.23 per cent of operations for adults and 41.9 per cent for children. It is the anesthetic of choice in surgery for children as it causes no damage to the inner organs. Since the introduction of evipan the percentage has decreased greatly in the case of operations on adults.

Rectal avertin was used for 2.75 per cent of adults and 9.75 per cent of children. It was generally combined with evipan, eunarkon or more rarely with chloroform, nitrous oxide or ether. For cleft palate and hare-lip operations in children avertin-chloroform anesthesia is preferred. The appearance of cyanosis or slow breathing which is due to an overdose is counteracted by injecting intravenously coramin until the toxic symptoms disappear.

Intravenous anesthesia with evipan is used for short operations. The anesthesia may be prolonged by the use of an inhalation anesthetic. It is contraindicated in toxic patients and fever.

Eunarkon, also used intravenously, does not differ markedly from evipan. Some patients become excited (5 per cent), and thrombosis was observed in some cases at the site of the injection.

Local anesthesia was used in 80 per cent of the adults; of these 23 per cent were cases of spinal anesthesia.

# Editorial

## Dr. R. Ottolengui

DR. R. OTTOLENGUI was born March 15, 1861, in Charleston, South Carolina. His grandfather, Dr. B. A. Rodrigues, was a dentist whose skill and personality extended his fame far beyond his native state of South Carolina. Dr. Rodrigues' preceptor was the historical figure, Dr. C. Starr Brewster, also of Charleston. Dentistry had background and indeed was traditional in the Ottolengui family.

From the biographic sketch of Dr. Ottolengui by Dr. J. R. Schwartz, we glean some of the high spots of Dr. Ottolengui's career: "At the Twentieth Anniversary of the Kings County Dental Society (celebrated in 1932) he was acclaimed an individual 'Possessing great intellectual attainments, keen and alert to the requirements of organized dentistry, he has through his deliberations and writings dispelled the fears, complexes and misunderstandings with which our profession has been so frequently confronted.' "

Very early in his career Dr. Ottolengui attracted the attention of "Dr. W. A. Atkinson, who at that time was known and considered as the dean of the dental profession. Undoubtedly, the heart of Dr. Atkinson was filled with kindness because his was an open door to young Ottolengui who was welcomed to bring his patients for advice and assistance. However, an ulterior motive and a beneficent one at that, was held out by Dr. Atkinson as a condition, that in after years as Dr. Ottolengui grew older, he should similarly open his office and give freely of his . . . experience to younger practitioners."

Dr. Ottolengui was one of the early pioneers in the practice of orthodontia. He also attracted the attention of Dr. Norman Kingsley in New York, who at that time was considered the most skillful of prosthetic workers, was a prominent orthodontist, as well as the originator of the most satisfactory method of treating cleft palates. "Dr. Kingsley recognized in him an apt pupil but before he took him under his wing, he also exacted from him a promise. He contended that the majority of cleft palate patients had little or no money but were nevertheless worthy of receiving service. In exchange for teaching his methods and technics to Dr. Ottolengui, his stipulation was merely that any patient requiring such treatment should receive this service regardless of any fee involved."

*Dental Items of Interest* had for years been his consuming passion. For almost forty years he conducted the famous round-table in that journal. In 1903 the newly formed American Society of Orthodontists requested the publication of their proceedings in *Dental Items of Interest.*

Dr. Ottolengui was a past-president of the American Society of Orthodontists and its outstanding nonspecializing orthodontist member. This is a distinction indeed, and ultimately was exemplified in the bestowing of honorary membership. In fact, honor upon honor was heaped upon him through-

out his active life—so many that space will not permit a complete listing here. Suffice it to say that probably no other dentist in America has received more honors or greater recognition during his lifetime than did Dr. Ottolengui.

During his early years he took a great interest in dental politics; he was a fluent speaker, endowed with ample courage and a "legal type" of mind. His powerful influence in the old National Dental Association was undisputed. Besides his accomplishments in dentistry, he had a wide range of other interests; he wrote fiction, did beautiful photography, and sculptured.

So passes another of those intrepid, enthusiastic, brilliant, personable and outstanding individuals who not only have helped carve out the destiny of American dentistry, but have contributed in no small way to the growth of the lusty infant, orthodontia. There were a number of energetic men (late in the last century and the early part of this century) who stood by at the birth of orthodontia, but the group has diminished rapidly during the last few years: Kingsley, Case, Angle, Ketcham, Dewey, Pullen, Watson, Young, Baker and a host of others, and now Ottolengui. His memory will linger and his important influence will be felt for all time in both dentistry and orthodontia.

*H. C. P.*

# News and Notes

## Orthodontic Directory of the World

The 1938 edition of the Orthodontic Directory of the World is now in the process of compilation. Those eligible for listing may communicate with the editor concerning same.

CLAUDE R. WOOD, Editor
Medical Arts Bldg.
Knoxville, Tenn.

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## Great Lakes Association of Orthodontists

The meeting of the Great Lakes Association will be held in Cleveland, November 1 and 2.

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## New Orthodontic Journal

A new orthodontia journal made its bow recently as the official organ of the Argentine (South America) Society of Orthodontists. The new journal contains 140 pages, and is well illustrated.

There are now three journals which devote the bulk of their pages to the specialty of orthodontia. That is interesting because no other department of dentistry supports such a rich literature. The Argentine journal will be a welcome addition to orthodontic literature and will no doubt be responsible for the attraction of wide interest in the subject among Spanish-speaking peoples.

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## Notes of Interest

Dr. A. Church announces the removal of his office to One East Forty-Second Street, New York City.

Dr. Brooks Bell announces the association of Dr. Tom M. Williams. Practice limited to orthodontia. Medical Arts Building, Dallas, Texas.

Dr. William M. Tweed announces the removal of his offices from the Luhrs Tower Building to 619 Professional Building, Phoenix, Arizona. Dr. Charles H. Tweed, consultant. Orthodontics exclusively.